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L'ECOLE D'AMEDEE MARIEN ET SES ELEVES RHEAUME ET PARE

EDOUARD DESJARDINS, M.D., F.R.C.S.[C], F.A.C.S.,* *Montréal, Qué.*

FAIRE revivre pour les générations actuelles et à venir la figure de certains maîtres de la chirurgie au Canada m'apparaît comme un devoir. Bien plus, quand il s'agit de parler du rôle joué à Montréal par Marien et ses élèves préférés Rhéaume et Paré, j'en fais plus qu'une obligation, je rends hommage et reconnaissance à trois maîtres.

Marien me renieait comme son élève, il est vrai, car je n'ai pas rempli la condition *sine qua non* pour le satisfaire, puisqu'il fallait à l'époque de mon internat passer douze mois consécutifs dans son service et qu'à ce même moment, 1921-22 et 1923, l'activité chirurgicale de Marien était en perte de vitesse.

Rhéaume n'était pas mécontent de me compter au nombre de ses assistants, bien que pour arriver au maître, il me fallait alors franchir l'obstacle d'un chirurgien senior qui s'interposait entre Rhéaume et les plus jeunes.

Paré m'a rendu le service inappréciable d'avoir su inspirer à ceux de ma génération la confiance en soi. Mieux que quiconque, Paré joignait la théorie à la pratique en nous permettant de mettre constamment la main à la pâte.

J'ai la conviction que le tryptique Marien-Rhéaume-Paré saura mettre en lumière la transposition à Montréal de la discipline chirurgicale de l'Ecole française, fin du XIXe et début du XXe siècle.

AMÉDÉE MARIEN

Marien est à Paris, peu après 1890, car après une brève incursion en pharmacie et en pratique rurale, il décide de faire de la chirurgie. Comme plusieurs collègues, il s'embarque pour la France; tous, suivant les mots de T. Parizeau, sont "pris d'un beau zèle—partis avec l'idée de se compléter aux sources mêmes de la science française, ils cinglèrent vers les côtes de France, décidés à ne revenir que l'esprit meublé d'acquisitions toutes fraîches et de notions à l'ordre du jour."[1]

La chirurgie française l'enthousiasme; il est l'élève de Legueu, de Quénu et de Lejars. Il s'éprend des réalisations de Terrier, de Péan et de Lucas-Championnière.

Fig. 1.—Marien, fondateur en 1897 du Comité d'études médicales, professeur de clinique et chirurgien en chef de l'Hôtel-Dieu de Montréal.

Marien (Fig. 1), de retour à l'Hôtel-Dieu de Montréal, cherche à se faire l'apôtre de la vérité nouvelle. Parizeau a les mots justes quand il dit: "Les premiers revenus de là-bas eurent à faire une vraie bataille avant de réussir à introniser chez nous les vues nouvelles, à faire accepter en haut lieu les méthodes de l'antisepsie. . . . Il fallut convaincre et ces chirurgiens et les administrations; ces dernières refusaient de permettre les frais additionnels de la chirurgie moderne. . . . Les serviettes et les éponges, après un simple nettoyage, pas-

*Professeur émérite de chirurgie à l'Université de Montréal et régistraire à l'Hôtel-Dieu de Montréal, Montréal, Qué.

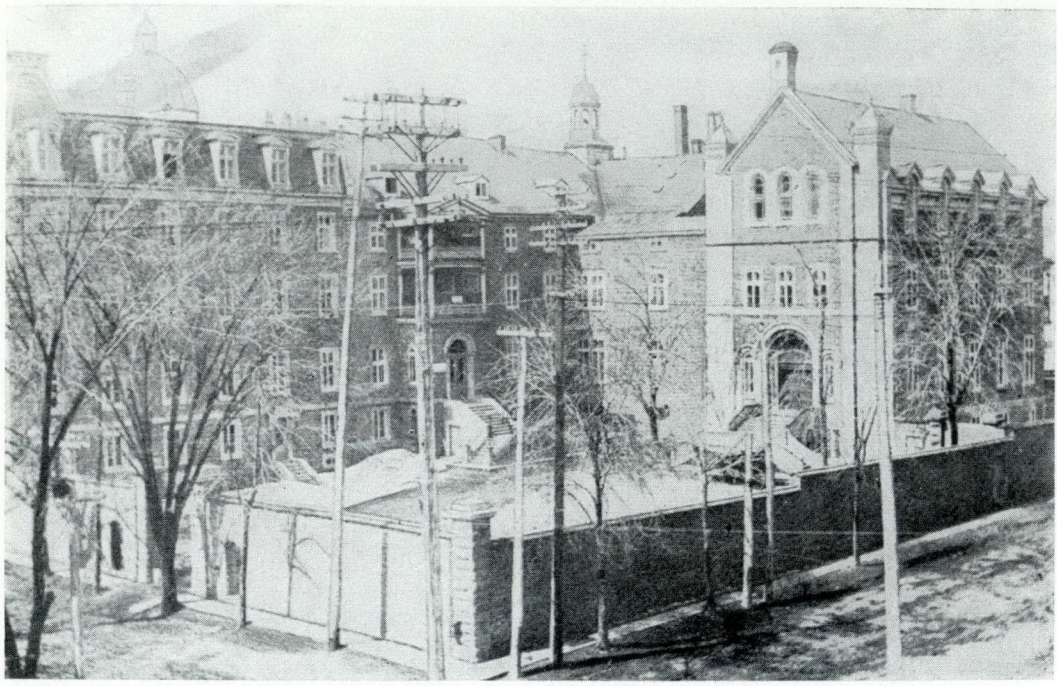

Fig. 2.—L'Hôtel-Dieu en 1901, à l'époque où Marien cherchait à introduire la chirurgie aseptique. L'aile de droite logeait au dernier étage les salles d'opération.

saient à d'autres plaies, les germes cueillis un peu partout. . . . Les théories nouvelles ne réveillaient que peu d'écho chez les anciens. . . . Rottot, Hingston et Brunelle pensaient de même, mais avec plus de modération peut-être que Brosseau."

"Les débuts de l'antisepsie furent difficiles à Montréal, ajoute Parizeau; j'ai souvenance que Marien dut plaider sérieusement pour qu'on lui permit d'installer tant bien que mal à côté de la salle d'opération ses propres appareils de stérilisation et pour qu'on voulût bien mettre en pratique les règles qu'il rapportait toutes fraîches des salles d'opérations de France [Figs. 2 et 3]. On peut admirer la belle énergie qu'il déploya dans la circonstance et les conséquences heureuses qui en découlèrent."

Marien revient de France, frappé des succès remarquables remportés là-bas dans les grandes cliniques chirurgicales par la méthode aseptique. Aussi voulut-il implanter les techniques idéales; ce qui ne se fit pas sans heurt.[2]

Si Marien avait tout de l'apôtre, s'il en manifestait l'enthousiasme et le zèle qu'aucun obstacle n'arrête, il n'avait rien du diplomate de carrière. Il parlait franc et dru, en langage clair et sans équivoque. Les compromissions subtiles, les dosages savants ne trouvaient jamais grâce chez lui. C'est dire que ce n'est pas facilement qu'il parvint à ses fins. Ses aînés n'aimaient pas l'idée que l'on pût bouleverser leurs procédures habituelles et ils excipaient de leurs résultats heureux pour se défendre d'une révolution gênante.[2]

Marien tient à l'Hôtel-Dieu le même rôle que Mercier remplit à Notre-Dame. L'un et l'autre comprennent la portée énorme des techniques opératoires nouvelles et l'intérêt primordial qu'en pourraient retirer les malades.

Son ami Dubé donne des détails savoureux: "Ceux de ma génération se souviennent très bien de l'insuffisante préparation des malades avant l'opération et aussi du grand nombre d'opérés qui ne guérissaient qu'après une longue suppuration." "Il me revient que l'interne de service d'alors préparait les ligatures qui devaient servir à l'opération en passant sur un bloc de cire du fil de chanvre, coupé en longueur définie et fixé avec soin sur le coin de la table près de l'opérateur. Je n'oublierai jamais non plus le pied en verre soutenant un

Fig. 3.—Une salle de malades (44 lits) à l'Hôtel-Dieu de 1861 à 1933.

coussin en forme de melon, avec ses tranches de velours de différentes couleurs, sur lequel étaient piquées les aiguilles qui devaient servir à l'opération. Les chirurgiens se bornaient à se laver soigneusement les mains et, une fois les ongles bien curés avec le coin de la serviette, avaient soin d'assujettir leur lorgnon avec les doigts qui l'instant d'après maniaient le bistouri. J'ai même vu un chirurgien, au cours d'une amputation de la cuisse qui présentait quelque difficulté, mettre son couteau entre ses dents, pour bien examiner, en s'aidant de ses deux mains, le fond de l'incision qu'il venait de pratiquer. Et pourtant! ces hommes de l'art avaient considérablement fait évoluer la chirurgie. Ils étaient de vrais enseigneurs, d'excellents techniciens."[3]

Marien est, à la salle d'opération, d'une discipline militaire. Ponctuel, il consacre à la préparation de son malade des soins très minutieux: rasage, lavage, désinfection; il se lave les mains avec méthode, car il connait les travaux de Halsted; il utilise pour leur désinfection, la plongée jusqu'au coude dans un bocal de permanganate de potassium, suivie d'un rinçage au bisulfite de soude.

Il exige le port du masque, difficile à cause des moustaches exubérantes, et celui des gants qui sont de coton blanc.

Marien opère à la française, très vite et bien; il excelle dans les amputations et les désarticulations. Sa dextérité est remarquable. Pour suivre la mode parisienne, il a un peu l'allure théatrale. Il ne plaisante pas à la salle d'opération; il commande sur un ton sans réplique; il lui faut, pronto, l'instrument réclamé.

Marien a des exigences post-opératoires. Dans la chambre de chacun de ses malades, l'infirmière doit installer ce qu'elle appelle: l'autel de Marien, soit une table recouverte d'un drap blanc, où sont alignés les bocaux stériles servant à contenir les instruments et les pièces de pansement. Un cathétérisme vésical fait à la Marien s'exécute selon une procédure aseptique rigoureuse.

La réputation de Marien est telle qu'un jour il doit se rendre à cheval à une vingtaine de milles de Montréal pour procéder à l'extraction d'une balle qu'un notable de l'endroit doit à l'exubérance colérique de son fils dénaturé. Les assistants ont la tâche d'emporter en voiture une table d'opération, des appareils à stérilisation, les instruments et les pièces de pansement.

A la mort de ses collègues Hingston et Brunelle, Marien fut nommé professeur de clinique chirurgicale par la Faculté de

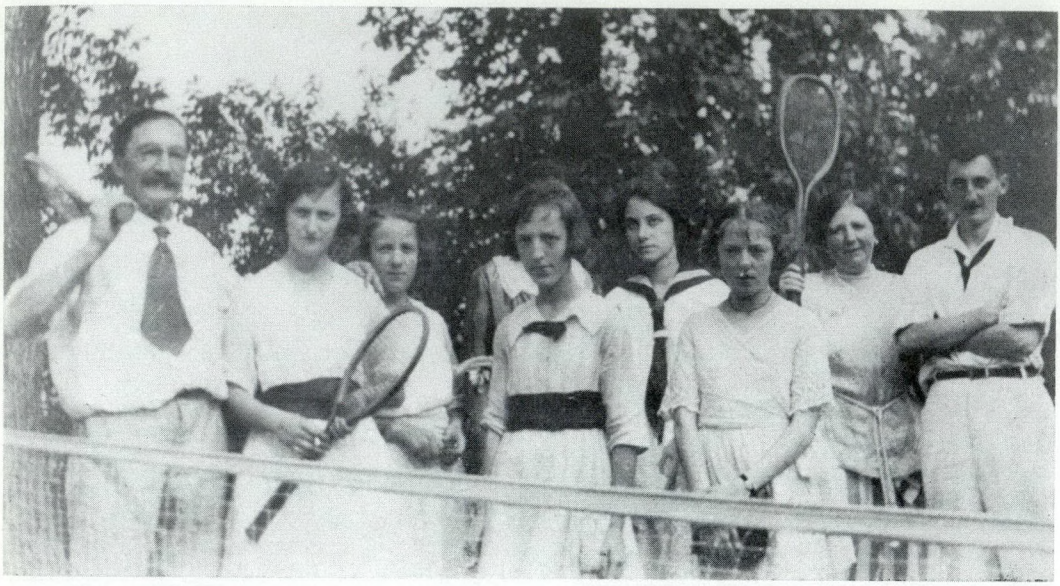

Fig. 4.—Marien, le chef rigide, vu dans un moment de détente, à son domaine de la Rivière-des-Prairies. Marien était dans l'intimité l'homme le plus courtois. A l'extrême droite, l'auteur de l'article (photo de l'été 1922).

Médecine et chirurgien en chef de l'Hôtel-Dieu. Excellent opérateur il était aussi un professeur méthodique.

Dubé dit que: "Marien se montra toujours très sévère aux examens de fin d'année. Il se rappelait ceux qui avaient été assidus à sa clinique; pour eux il était rempli d'aménité, mais il se montrait impitoyable pour les élèves qui n'avaient pas été ponctuels dans son service."[3]

La réputation de Marien dépassa vite les bornes de la Cité; on venait de partout le consulter. Sa maîtrise opératoire faisait l'orgueil de ses élèves et l'admiration des médecins étrangers. La réputation de Marien en tant qu'homme était diversement appréciée. On l'abordait avec timidité, car il était d'approches difficiles. Il avait un regard pénétrant que magnifiait le port de verres épais. Sourcils poussant dru et grosses moustaches menaçantes n'étaient pas de nature à adoucir ses traits. Marien savait auprès de ses malades se faire doux et bienveillant; il était à son domaine de la Rivière-des-Prairies d'une grande hospitalité et d'une politesse extrême (Fig. 4).

A l'hôpital, il n'était pas toujours patient et il lui arrivait, si une faute était commise, de déclencher une colère formidable qui effrayait assistants et gardes-malades; il ne tardait pas cependant à faire un prompt retour sur lui-même; il s'excusait alors de sa vivacité et regrettait vivement son manque de modération. Les internes ne l'abordaient pas sans trembler et cependant il était d'une mansuétude extrême pour celui qui savait trouver les accents capables de l'émouvoir; la recette était simple; il suffisait d'être poli, respectueux et clair dans ses propos.

"Marien avait également des traits de ressemblance avec son aïeul, le Dr Amable Simard, dont l'originalité et les sautes d'humeur ont été racontées par ses contemporains. Ce vieux médecin était de bonne taille, vigoureux et soigneux de sa personne. Il aimait à porter costume blanc et faire, à cheval, ses visites de malades. Les amis de Marien se rappellent qu'il était lui aussi très soigneux de sa personne et qu'il affectionnait tout particulièrement les longues randonnées à cheval, à la campagne.

Marien avait toujours été d'une robustesse remarquable. Sans être de haute taille, il avait la carrure et les muscles d'un athlète. Il était infatigable. Sur sa ferme, il savait aussi bien labourer, faire les semailles et la moisson que dompter les jeunes pur sang qu'il élevait, chaque fois que son fermier s'en déclarait incapable. Une fois en selle Marien y restait, coûte que coûte et devenait maître de sa monture."[3]

Marien participe aux Congrès tenus à Montréal. Il écrit beaucoup dans l'Union Médicale du Canada de 1896 à 1922, mais depuis, des troubles oculaires l'obligent à diminuer son apport opératoire et à compter de plus en plus sur l'assistance majeure de son élève et neveu Armand Paré.

Marien souligne dans une conférence l'importance de la régularisation des cadres chirurgicaux: "Ne devrait-il pas y avoir une corporation spéciale des chirurgiens? Cette corporation serait soumise à un code très sévère qui assurerait la compétence et l'honorabilité de ses membres."[3] Les idées germent. Il ajoute dans une autre circonstance: "Dans les grands mouvements du socialisme moderne chez toutes les classes de la Société, les professions et les associations ne s'organisent-elles pas pour s'entr'aider, se soutenir et améliorer le sort de leurs membres?"

Il faut près d'un demi-siècle avant que ne se réalisent, grâce à la fondation de l'Association des Chirurgiens de la Province de Québec, ces paroles d'espérance: "Si les idées que j'émets pouvaient un jour arriver à une réalisation, je m'estimerais heureux d'y avoir contribué."[4]

Marien, conscient de ce qu'il devait à la France, fit des efforts désespérés pour organiser un hôpital militaire en 1914; il offrit ses services de chirurgien sans solde. L'âge, l'importance de son travail à l'Hôtel-Dieu s'y opposent. Ce rêve, ses élèves Rhéaume et Paré le réalisèrent.

Pierre-Zéphir Rhéaume

Rhéaume (Fig. 5) est tout le portrait de Marien; même formation à Paris, mais dix ans plus tard; installation première à Valleyfield; retour à l'Hôtel-Dieu; service chirurgical actif, carrière professorale accomplie avec méthode; initiation des internes et des étudiants à la chirurgie opératoire par des séances à l'Hôtel-Dieu; enrôlement dès sa formation dans l'Hôpital Général Canadien no. 6 de l'Université Laval de Montréal au poste de chef de chirurgie; chirurgie militaire intense à Troyes, puis à Joinville-le-Pont; reprise, la paix signée en 1918, de la clientèle; publication d'un volume sur la chirurgie de l'estomac et du duodénum, publié à Paris chez Masson et Cie; organisation de l'hôpital Saint-Luc renové; mise en fonction de l'organisme directeur de l'Association des Médecins de langue française du Canada.

Fig. 5.—Rhéaume, ancien lieutenant-colonel du Corps expéditionnaire médical canadien, professeur de médecine opératoire, chirurgien en chef de l'Hôpital Saint-Luc de Montréal.

Les limites restreintes de ce tableau exigent une énumération lapidaire des titres et travaux de Rhéaume.

La gratitude que je dois à Rhéaume me porte à l'occasion de sa mort, à faire de sa carrière un tracé peut-être infidèle et injuste —ces mots d'alors, je les pense encore, aujourd'hui. Il m'ouvrit, sans sollicitation de sa part les portes de son service; il le fit, en grognant, d'un ton qu'il voulait bourru, souvenir de guerre sans doute. Il a la mime de l'ogre, quand on lui décerne un éloge ou lorsqu'on esquisse un mot de remerciement.

Habitué au commandement militaire, il a le langage vert et bref. Sans malice, il émaille ses propos d'interjections peu orthodoxes. Il faut regarder l'œil de celui qui les prononce; il n'est ni perfide, ni méchant, mais gouailleur. Rhéaume imite alors ses patrons de Paris: Kirmisson, Edouard Quénu, Broca ou Legueu.

Très actif, Rhéaume ne boude pas à l'ouvrage; il devient la cheville ouvrière du Bulletin de l'A.M.L.F.C., et il collabore régulièrement à l'Union Médicale du Canada.

A l'étroit dans le cadre restreint de son service à l'Hôtel-Dieu, il entrevoit, à Saint-Luc, l'espace vital rêvé.

Il quitte en 1930 l'Hôtel-Dieu pour devenir le grand patron du nouveau Saint-Luc, où il attire une pléiade de jeunes.

L'Ecole française, vue d'abord sous son uniforme civil de fin de siècle, puis vécue dans son amplitude à l'arrière front des armées de 1915 à 1918, imprègne fortement sa personnalité; la ponctualité, l'asepsie rigoureuse, la présence post-opératoire exigée des assistants auprès des grands malades, la tenue des dossiers sont des règlements qu'il ne faut pas enfreindre dans le service de Rhéaume. Il s'intéresse beaucoup à la chirurgie de l'estomac et de l'intestin; il opère vite, car il connaît l'anatomie topographique à fond; bien qu'il essaye de le prévenir, il ne comprend pas bien le mécanisme déclenché par l'agression chirurgicale et la traction qu'il fait sur les mésos fait souvent bondir de désespoir son anesthésiste préféré, Charles LaRocque.

Rhéaume a horreur de la chirurgie faite du bout des doigts; il refuse parfois de prolonger un temps opératoire pour ne pas céder à la tendance des "chichis" et de la chirurgie de dentellière.

Rhéaume est sur certains points le digne émule de son maître Marien et il sert de modèle à son disciple Armand Paré.

Armand Paré

Armand Paré (Fig. 6), neveu de Marien, est l'image parfaite morale, intellectuelle et professionnelle de Rhéaume; même formation, même apprentissage, mêmes patrons en France, carrière militaire presqu'identique, apport à l'enseignement similaire, pratique chirurgicale apprise à la même école et qui se déroule à la même cadence.[5]

L'émotion, que j'ai ressentie à la mort de mon maître et ami Armand Paré m'a inspiré les mots de gratitude qui s'imposait et la méditation m'a permis de revoir en pensée le Paré que j'ai décrit et je ne peux rien ajouter à ce que j'écrivis en mai 1956.

Energique, véritable héros des tragédies grecques, il refuse le repos; il continue à opérer avec une fracture de la clavicule; irrémédiablement touché dans ses forces vives, il ne s'accorde aucune minute de détente.

Fig. 6.—Armand Paré, chirurgien de l'Hôtel-Dieu, ancien major de l'Armée canadienne, déteste faire parler de lui. Cette photo le représente dans un moment de détente; Paré a semblé ne jamais vieillir et même à la veille de sa mort, il paraissait encore jeune.

Travailleur infatigable, le premier à l'œuvre, Paré a la passion de son métier et trouve la plénitude de son bonheur à la salle d'opération. La mort implacable ne lui a pas donné la consolation souhaitée de mourir à la tâche, sur la brèche, au travail, debout, les armes à la main; il s'en est fallu de peu, puisqu'à peine 12 heures avant sa fin, il fait deux interventions laborieuses et qu'une gastrectomie est inscrite au tableau pour le matin même de son grand départ.

Fils et petit-fils de médecins, neveu de chirurgien, Paré est élevé dans l'ambiance médicale et tôt se destine à la carrière chirurgicale. Il s'enrôle dès 1915 dans l'armée impériale, pour être peu après versé dans le corps médical canadien. A sa demande, on l'envoye sur la ligne de feu avec la 11e Ambulance canadienne de campagne. Il y gagne la Military Cross et la Croix de Guerre française avec étoile de bronze et citation du maréchal Pétain. Voici un extrait de la citation: "L'officier médical Armand Paré a fait preuve du plus grand dévouement vis-à-vis de la population civile d'Anzin, notamment pendant les journées du 27 au 30 octobre, où il s'est dépensé sans compter pour soigner les malades,

blessés ou intoxiqués, transportant lui-même sous un violent bombardement, en plein tir des mitrailleuses, pour les mettre en sécurité et leur prodiguer ses soins, les civils impotents qui ne pouvaient arriver à franchir les clôtures. A sauvé ainsi, en risquant sa propre existence, un grand nombre de vies françaises. Au grand quartier général, le 10 avril 1919, le maréchal de France, P. Pétain."

Paré n'a jamais dévié; tel il s'est montré durant la guerre, tel il a vécu depuis sans jamais se démentir. Son meilleur titre de gloire est d'avoir su contribuer à la formation chirurgicale de nombreux élèves. Il est le patron, parfois bourru, sarcastique ou sévère; un patron qui souligne sans lourdeur la faute et qui indique le remède; un patron qui a horreur des gestes inutiles, mais qui ne marchande pas ses conseils et qui ne croit pas déchoir en assistant lui-même ses élèves.

Sous des dehors brusques, avec des gestes à l'emporte-pièce, Paré cache soigneusement un cœur d'or; il est d'une sensibilité exquise et il reste bouleversé quand son tempérament vif le pousse à dire des paroles désagréables.

Humaniste, Paré est amoureux des lettres; il lit et sans répit. Il respecte les articles bien faits et il méprise le peu de respect pour la syntaxe et l'orthographe. La lecture le passionne au point qu'il ne veut perdre aucun instant et qu'il lit même en marchant de chez lui à l'hôpital.

Doué d'un beau talent littéraire, il eut enrichi notre patrimoine; malheureusement, timidité ou complexe inexplicable, il ne publie qu'un seul article dans toute sa carrière; article par ailleurs remarquable et qui parut dans le Journal de l'Hôtel-Dieu, en janvier 1933.

Pionnier de la chirurgie abdominale à l'Hôtel-Dieu, expert en gastrectomie, M. Paré est un opérateur rapide et certain de ses gestes. Anatomiste accompli, il ne perd pas de temps à la ligature des capillaires. Philosophe, disciple de Lecène et de Leriche, il a compris, durant son demi-siècle de métier, qu'en chirurgie comme en histoire, la vie est un éternel recommencement. Une mode nouvelle est le rappel, sans mention des sources, d'une méthode ancienne passagèrement mise sous le boisseau.

A la manière de Marien et de Rhéaume, sa vie est simple; lever à l'aube, travail ardu, quotidien, sans relâche, détente à la lecture de ses auteurs préférés et pendant les vacances, promenade sur son bateau. En résumé, Paré est une figure remarquable, un caractère trempé à l'antique, un homme de devoir et un chrétien stoïque.

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ORIGINAL ARTICLES

NON-PENETRATING THORACIC TRAUMA: A STATISTICAL SURVEY AND ANALYSIS*

DENIS S. DRUMMOND, M.D., ROSS H. CRAIG, M.D. and EDWARD DOW, M.D., *Ottawa, Ont.*

In civilian practice, thoracic trauma is an ever-increasing problem. In 1958, 25% of deaths due to automobile accidents in the United States were directly associated with trauma to the chest.[1, 2]

Although the mortality rate in penetrating chest injuries has shown a definite decrease in recent years, there has been no dramatic improvement in survival among those patients with crushing trauma to the chest. Various workers[1, 3] have found that the mortality rate for non-penetrating chest injuries is about four times higher than that for penetrating injuries.

The following communication describes our experience with blunt chest trauma at the Ottawa Civic Hospital. It is a statistical survey of non-penetrating injuries admitted to this hospital during the five-year period beginning January 1, 1960 and ending December 31, 1964.

The survey covered 194 cases. An attempt was made to determine the etiology and nature of the injuries, as well as the results and complications of their management.

There has been some selection of the cases. An effort was made to exclude those patients who had minor chest trauma. Only those cases have been selected in which three or more ribs or the sternum were fractured, or where there was some injury to the soft tissues such as the lungs, heart and pleura.

For the sake of convenience and clarity, the series has been divided into two broad groups:

Group I.—Uncomplicated chest injuries
Group II.—Complicated chest injuries
(a) complicated stable
(b) complicated unstable ("flail" chests)
(c) miscellaneous.

Group I.—Uncomplicated Chest Injuries

This group includes stable fractures to the thoracic cage without major soft-tissue injury of the chest. One hundred and sixteen of the 194 cases of blunt trauma were uncomplicated injuries.

Automobile accidents were the most common cause of injury in Group I—60% (Table I). In the more serious injuries of Group II, however, an even greater percentage (72%) were due to road accidents.

TABLE I.—Cause of Trauma in 116 Uncomplicated Chest Injuries

Cause	*No. of patients*	*%*
Automobile	70	60.3
Falls	36	31.1
Other (athletic, etc.)	10	8.6
Total	116	100.0

Most of the uncomplicated chest injuries were treated conservatively with analgesics, strapping of the chest, local anesthetic infiltrations or with bed rest alone. The complication rate was low. There were two fatalities—a mortality rate of 1.7%. One patient died of bronchopneumonia; the other died from a head injury. The corrected hospital stay* was 10 days.

Group II.—Complicated Chest Injuries

Patients with complicated chest trauma had a moderate to extensive amount of soft-tissue damage as well as bony injury to the chest. A breakdown of these cases is outlined in Table II. The miscellaneous cases from Table II will not be discussed further; there were no deaths in this group.

*From the Department of Surgery, Ottawa Civic Hospital, Ottawa, Ont.

*Calculated by omitting long stays in hospital due to other factors (e.g. fractured femur).

TABLE II.—Complicated Chest Trauma

	No. of cases
(a) Complicated stable	65
pneumothorax	
hemothorax	
other (i.e. lung contusion)	
(b) Complicated unstable ("flail" chest)	13
(c) Miscellaneous	9
ruptured diaphragm 4	
ruptured trachea 2	
ruptured bronchus 2	
pericardial tear 1	

Complicated Stable Chest Injuries (Table II)

There were 65 patients with complicated stable chest injuries; most of these had a pneumothorax, hemothorax or both. Some patients suffered local or diffuse hemorrhage and edema of lung parenchyma. This condition is commonly called lung contusion or traumatic "wet lung". Others had myocardial contusion. These cases were often associated with injury to other organ systems. The complication rate was higher than among patients in Group I, and included pneumonia, empyema, persisting hemothorax and fibrothorax.

There were five deaths among the 65 cases, a mortality rate of 7.7%.

Table III shows the cause of the trauma in the complicated, stable group. Automobile accidents accounted for 66% of the injuries.

Figure 1 shows the distribution of the injuries in the 65 patients in this group. Virtually all had multiple fractures to the ribs, sternum or both. Fourteen had pneumothorax alone and 18 had hemothorax alone; most, however, had both pneumothorax and hemothorax (31 cases). The treatment of pneumothorax in these 31 patients is considered separately under pneumothorax and hemothorax (Fig. 2).

Pneumothorax.—In this group of stable chest injuries, 45 patients with pneumothorax were treated: three by thoracentesis; 24 by continuous underwater-seal drainage; and four by thoracotomy for repair of a lacerated lung. The remainder (16 cases) were treated conservatively without any form of drainage. Most of those treated conservatively had a small pneumothorax with less than 20% collapse of the lung.

TABLE III.—Cause of Trauma in Complicated Stable Group

Cause	*No. of patients*	%
Automobile	43	66.2
Falls	18	27.8
Other	4	6.0
Total	65	100.0

The outcome in these 45 patients with pneumothorax was fairly good, regardless of the type of treatment. Most patients had uneventful recoveries with few sequelae. There were two short-term complications—both bronchopneumonia. Both patients survived. There were *no* long-term complications.

Hemothorax.—In the complicated, stable group (Fig. 2), 49 patients were treated for hemothorax: 23 by thoracentesis requiring

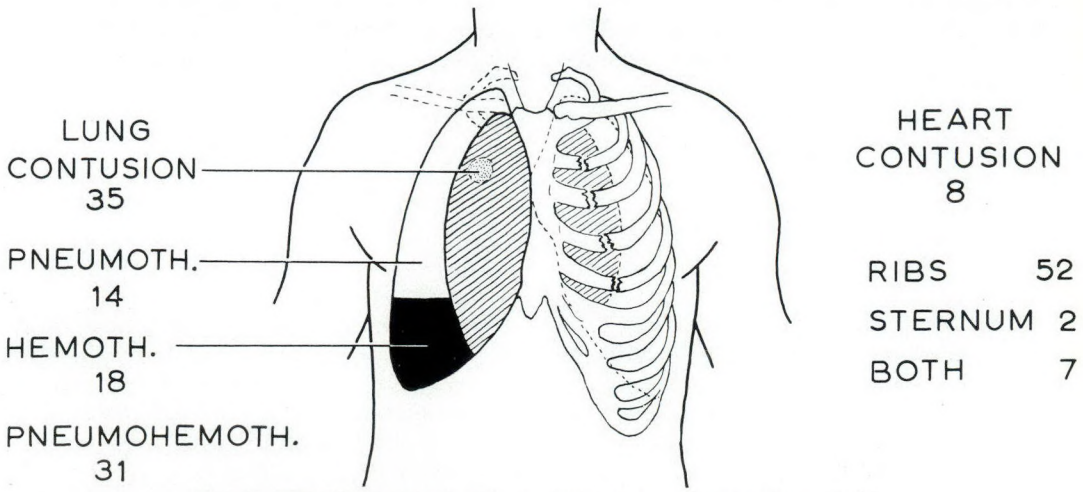

Fig. 1.—Distribution of injuries in 65 patients: five fatalities—7.7%.

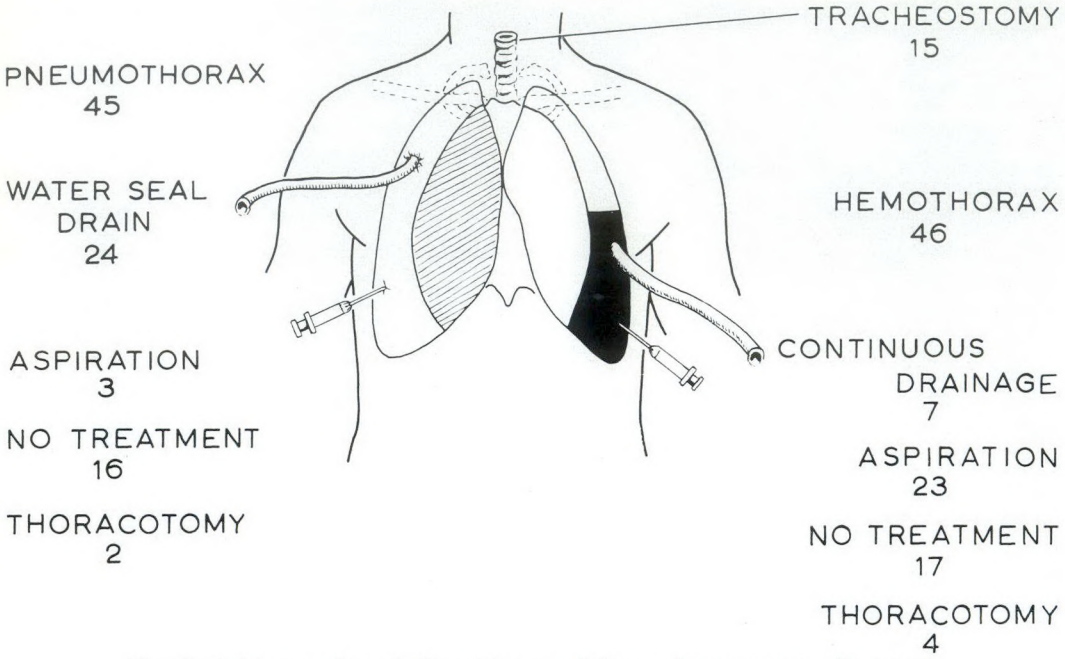

Fig. 2.—Stable complicated chest injuries and forms of treatment in 49 patients.

one to many aspirations; seven had thoracostomy and continuous tube drainage with or without suction; and four had a thoracotomy to control bleeding. Seventeen were treated conservatively without any form of drainage. Most of these had a small hemothorax.

More complications were encountered in the patients with hemothorax than in those with pneumothorax. There were eight major complications; two were short-term bronchopneumonia, and bronchopneumonia with congestive heart failure. However, six patients had long-term complications (there were none in the pneumothorax group). One patient developed empyema; two with persisting hemothorax were lost to follow-up; and three had major fibrothorax preventing lung expansion. Two cases of fibrothorax were due to persistent and organized hemothorax and one followed empyema. In three patients, thoracotomy and decortication was performed.

All of these six patients with long-term complications had been treated by multiple thoracenteses and none by continuous chest drainage. No complications were encountered among the seven patients treated by thoracostomy and drainage.

Much controversy surrounds the management of hemothorax. Although some authorities[4] advocate early aggressive treatment, many[5, 6] treat these patients conservatively by observing them and performing thoracentesis when it seems indicated.

No firm conclusions can be drawn from a survey as small as that reported in this communication; however, it would seem that the patients in our series with a large hemothorax were treated more successfully with continuous chest drainage than by multiple aspirations. In addition, continuous chest drainage is valuable in assessing persistent intrapleural bleeding and the need for blood replacement. Continuous chest drainage should be seriously considered in the presence of moderate to extensive hemothorax.

Complicated Unstable Chest Injuries (Table II)

Thirteen patients had "flail" chest: 6.7% of the 194 cases.

Besides the instability of the chest, most of these patients had considerable parenchymal injury such as traumatic "wet lung", pneumohemothorax and heart contusion. In all 13, the trauma resulted from automobile accidents.

Eight patients had multiple injuries out-

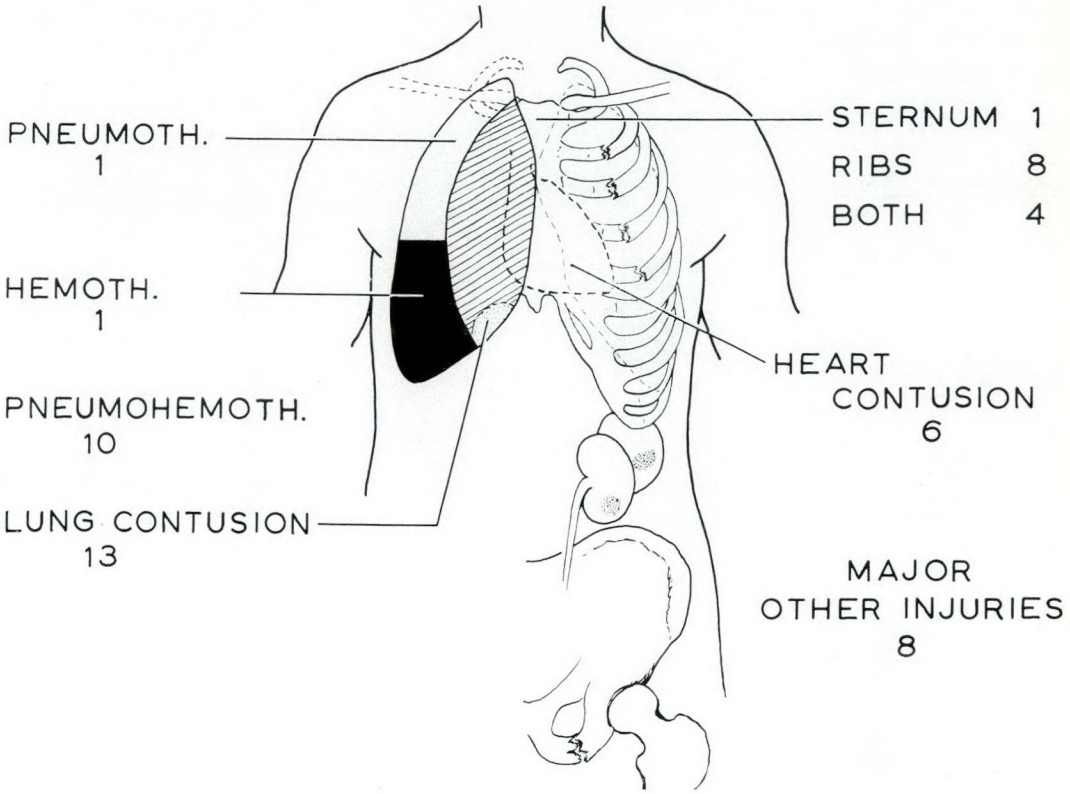

Fig. 3.—Unstable complicated chest injuries in 13 patients: seven fatalities—54.2%.

side the chest including head and abdominal injuries, and fractures to the long bones and pelvis (Fig. 3).

Twelve patients had multiple fractures to the bony thorax and one had a comminuted fracture of the sternum. Most significant was the amount of parenchymal injury within the chest. All 13 had associated lung contusion or traumatic "wet lung", and 12 had pneumohemothorax. Six patients had electrocardiographic or autopsy evidence of myocardial contusion.

Figure 4 shows the treatment employed in these unstable chest injuries. Some kind of chest stabilization was instituted in 11 patients. Traction through the sternum or ribs was used in about one-half of these patients, and stabilization by open reduction and fixation was used in five. Three patients had a thoracotomy for repair of lung lacerations. Ten patients had a tracheostomy and eight of these required positive-pressure respiration (PPB), either intermittently or continuously. Continuous chest drainage was carried out in 10 patients and thoracentesis in three. Antibiotics were administered prophylactically in 12 of the 13 patients.

The morbidity and mortality was high; there were seven deaths—a mortality rate of 54%. Of these seven, the chest injury was thought to be the principal cause of death in six and a major contributing cause in the other.

All fatal cases went to autopsy. Massive contusion of the lung with hemorrhagic consolidation was found in all. In three, subendocardial hemorrhages were seen and in one, a bloody pericardial effusion.

The earliest death occurred on the second hospital day. The longest survival was 13 days. In the patient who died on the second day, the extent of his injuries went unrecognized until shortly before death. Therefore treatment was inadequate and late. If this case is excluded, death occurred, on the average, on the ninth hospital day.

Among the six survivors in the complicated unstable group, the average hospital

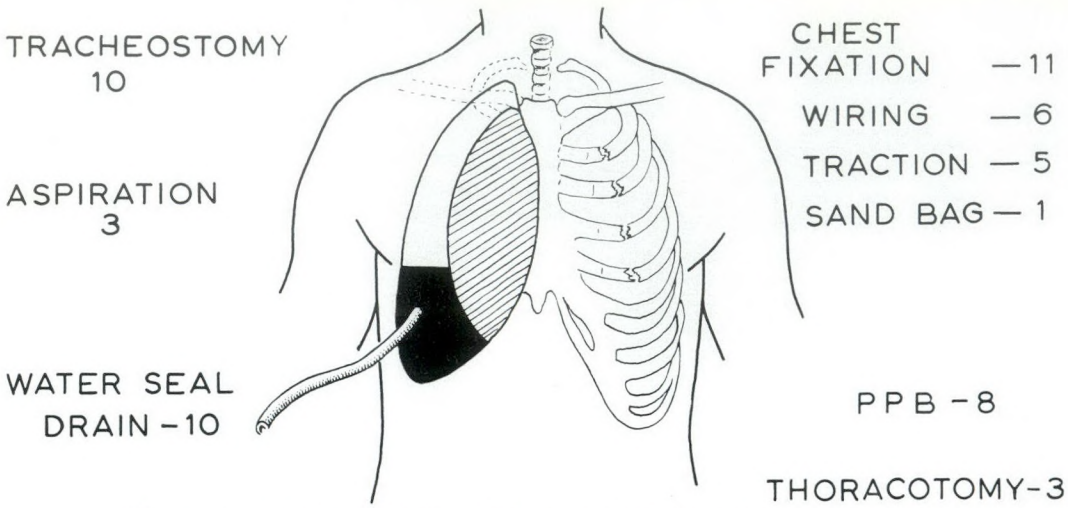

Fig. 4.—Treatment employed in unstable complicated chest injuries in 13 patients.

stay was 49 days. One serious complication developed that required long-term treatment, i.e. fibrothorax secondary to organized hemothorax. As noted earlier, the hemothorax had been treated by multiple thoracenteses rather than by continuous tube drainage. This, the fourth case requiring late decortication, reaffirms our earlier observations on the management of large hemothorax.

In most of these patients with unstable chest injuries, the clinical courses followed a typical pattern. Often clinical improvement was seen after early aggressive treatment but typically deterioration occurred towards the end of the first week. Chests that had previously been stabilized began to "flail" again. Bronchopneumonia was a frequent complication (eight cases) as was congestive heart failure. Tachypnea, cyanosis and hypercapnea marked a progressive downhill course.

Discussion

In this study, the patient's clinical course varied with the extent of lung contusion. Traumatic "wet lung" or "stiff-lung syndrome" is a poorly understood entity. The increased stiffness observed in this condition reflects the decreased lung compliance and may be due to pulmonary edema and a widespread patchy atelectasis caused by obstruction of the small bronchi with secretions.

Chest stabilization and tracheostomy with frequent suctioning have been the basis of treatment. More recently respirators have been employed.

The present series is too small to permit evaluation of the different types of chest fixation and the efficacy of positive-pressure breathing cannot be assessed. Other workers,[7, 8] however, have achieved an improved survival by the use of a piston-type respirator. In outlining its use, Avery, Mörch and Benson[7] draw attention to the value of the respirator both in preventing atelectasis and in altering negative intrapleural pressures, so that the "flail" segment can move with the thorax in inspiration rather than paradoxically. Ransdell[8] in his study reduced the mortality rate from 56% to 45% with the use of a piston respirator, employed either continuously or intermittently. Other workers have reported similar encouraging results but the mortality rates remain high.

The recent literature suggests that frequently traumatic "wet lung" is aggravated by an increase in catecholamine production stimulated by a low pO_2, a rising pCO_2 and acidosis. Precapillary vasoconstriction ensues, preventing blood from entering the microcirculation. The already embarrassed lung then has to cope with a greater circulatory load and an increase of interstitial fluid.[8] Adrenergic blockade has recently been advocated by Gurd to abolish the catecholamine effect and redistribute the intravascular volume to the microcircula-

tion. He reports two cases where pulmonary edema was successfully cleared by the use of phenoxybenzamine.[8]

Ransdell believes that secondary bronchopneumonia is responsible for the high mortality of traumatic "wet lung". Although many advocate the prophylactic use of antibiotics,[9] there is no clear evidence of benefit from them. Antibiotics were employed prophylactically in 12 of our patients; however, this did not prevent infection because bronchopneumonia developed in eight of them.

Although much work has been done in the last decade with respect to traumatic "wet lung", the mortality rate remains appallingly high. This can perhaps be changed in the future by the combined use of hyperbaric oxygen and piston respirators. The hyperbaric chamber could improve oxygenation despite lung damage, while the respirator would assist in CO_2 elimination. These measures along with supplementary aids such as adrenergic blockade may improve survival.

Summary

A review of 194 cases of blunt chest trauma admitted to the Ottawa Civic Hospital during the years 1959 to 1964 (inclusive) has been presented. These cases were classified into broad groups based on the presence or absence of associated parenchymal injury and the stability of the thoracic cage. The etiology, nature of the injuries, treatment and complications were evaluated.

Trauma secondary to automobile accidents was the causative factor in 65% of these cases.

In our series, hemothorax proved more difficult to treat than pneumothorax.

In this series, hemothorax was more effectively treated by continuous chest drainage than by multiple thoracenteses. A plea is made for more aggressive management of major hemothorax with thoracostomy and continuous tube drainage.

Experience with 13 patients with "flail" chest was analyzed. The mortality and morbidity was high and varied directly with the amount of lung contusion. A short discussion of traumatic "wet lung" was presented.

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Résumé

Les auteurs ont passé en revue 194 cas de traumatisme thoracique contondant qui se sont présentés à l'Hôpital Civique d'Ottawa. Ces cas ont été groupés comme suit: (1) blessures de la cage thoracique, (2) blessures graves du parenchyme à l'intérieur de la cage thoracique, et (3) thorax en "fléau".

Soixante-cinq pour cent de ces cas ont été causés par des accidents d'automobile. La mortalité globale a été de 7.2%.

On n'a éprouvé aucune difficulté à traiter les 45 malades de cette série de blessés qui avaient un pneumothorax. Toutes les méthodes thérapeutiques ont également réussi.

Quant aux 49 malades qui présentaient un hémothorax, le traitement par thoracostomie et drainage a été préférable que celui comportant de multiples thoracocentèses. La thoracostomie et le drainage n'ont entraîné aucune complication. Chez six des 23 malades traités par aspirations, on a observé des complications à long-terme: empyème (un cas), hémothorax persistant (deux cas) et fibrothorax ayant exigé la rupture des adhérences (trois cas). Le traitement du gros hémothorax doit être énergique et complet.

Treize cas de thorax en "fléau" ont été passés en revue. La mortalité a été élevée (54%) et était directement proportionnelle avec la gravité de la contusion pulmonaire. La bronchopneumonie, séquelle fréquente, a été observée dans huit cas. L'emploi prophylactiques d'antibiotiques n'a pas amélioré le traitement de ces cas. Les auteurs exposent les traitements classiques et les progrès thérapeutiques récents, notamment l'emploi de respirateurs à piston et le blocage adrénergique dans la contusion pulmonaire avec épanchement, d'origine traumatique.

THE UMBILICOPORTAL APPROACH FOR THE STUDY OF SPLANCHNIC CIRCULATION: TECHNICAL, RADIOLOGICAL AND HEMODYNAMIC CONSIDERATIONS

P. LAVOIE, M.D., F.R.C.S.[C], F.A.C.S.,* M. JACOB, M.D.,† J. LEDUC, M.D., Ph.D.,‡ A. LEGARE, M.D.§ and A. VIALLET, M.D., F.R.C P.[C],¶ *Montreal, Que.*

In January 1964, Bayly and Carbalhaes[1] reported a new approach to the study of the portal system using the umbilical vein|| for cannulation of the left branch of the portal vein.

A preliminary report of this procedure was previously presented in this journal.[2] A midline extraperitoneal incision 5 cm. long is made under local or general anesthesia and the round ligament of the liver, located between the leaflets of the falciform ligament, is catheterized directly.

This technique had certain serious limitations, however, providing at best, opacification of the liver parenchyma, and only in exceptional cases retrograde injection of the portal vein and collateral circulation—the essential object of radiologic studies in portal hypertension.

With exact positioning of the catheter, it is now possible to perform selective splenic, mesenteric or portal venograms. This procedure, as we now carry it out (*vide infra*), appears to be a real improvement, since other workers using this method have apparently not yet succeeded in achieving constant opacification of the portal vein.[1, 3]

Since our preliminary report, we have carried out this improved procedure under local and/or epidural anesthesia with satisfactory results in 34 patients, whose diagnoses are shown in Table I.

The purpose of the present communication is threefold: (1) to describe further technical advances in this procedure; (2) to present our latest radiologic results; and (3) to present some preliminary hemodynamic and biochemical data from eight patients with portal hypertension.

Technical Comments

In the preliminary report,[2] our findings in 60 cases were presented. Initially, at autopsy, the umbilicoportal junction was carefully dissected out in 10 cadavers in order to verify that the round ligament was patent in adults; it was found that this patency was constant. Soon thereafter, the round ligament was catheterized in 50 patients before or during laparotomy. Personal experience with the procedure was thus acquired and the efficiency of this technique in the diagnosis of portal hypertension, cirrhosis or metastatic liver disease was frequently shown.

Anesthesia

The cannulations in our last 34 patients, reported in the present communication, were done without general anesthesia. At first, the abdominal wall was infiltrated with local anesthetic, but the degree of analgesia so obtained was not sufficient. Necessary traction on the ligamentum teres during catheterization and the injection of

TABLE I.—Diagnoses in 34 Patients Undergoing Umbilicoportography

Diagnoses	*No. of patients*	*Total*
Cirrhosis Laennec's	7	
Postnecrotic	3	10
Alcoholic hepatitis	3	3
Gastrointestinal neoplasm without hepatic metastases	10	10
Hepatic metastases from cancer of		
stomach	3	
colon	2	
rectum	3	
kidney	1	
breast	1	10
Hepatoma (superimposed on postnecrotic cirrhosis)	1	1
		34

*Surgeon, Hôpital St-Luc, Montreal, and Assistant Professor of Surgery, University of Montreal, Montreal, Que.
†Anesthesiologist, Hôpital St-Luc.
‡Assistant Professor of Physiology, University of Montreal.
§Radiologist, Hôpital St-Luc, and Assistant Professor of Radiology, University of Montreal.
¶Physician, Hôpital St-Luc, and Assistant Professor of Medicine, University of Montreal.
||The umbilical vein passes from the umbilicus to the left branch of the portal vein, occupying the free border of the falciform ligament. After birth, the umbilical vein becomes a fibrous cord—the round ligament (ligamentum teres) of the liver. This structure is not occluded but is still patent in adults, and can be catheterized inside the liver up to the portal vein.

radio-opaque material under pressure into the portal vein were often painful.

We now prefer continuous peridural anesthesia. The catheter used for anesthesia is left in the peridural space and this permits easy control of the spread and timing of analgesia. It is started before the catheterization, allowed to fade during the recording of pressures or any sampling under basal conditions and brought back again for the injection of radio-opaque material.

Even though peridural anesthesia abolishes all sympathetic and somatic pain during the procedure, the patient still feels minor discomfort, which is believed to be due to parasympathetic activity.[4]

This method permits the recording of hemodynamic data under basal conditions and the whole procedure is done under satisfactory analgesia.

Operative Technique

The ligamentum teres is reached extraperitoneally, advancing between the two thin leaflets of the falciform ligament, as described previously.[2] Formerly, to achieve exact positioning, it was necessary to open the peritoneal cavity and direct the catheter carefully into the portal vein, holding it pinched between the thumb and the index finger.

When this delicate maneuver failed, the alternative was to inject the radio-opaque material under high pressure into the left branch of the portal vein, in an attempt to obtain a complete retrograde portosplanchnogram (Fig. 1). However, it soon became obvious that this approach did not produce consistent or reliable results, because, even when hepatograms were done regularly and detected metastases in the liver, retrograde opacification was usually not sufficient to delineate the portal vein and collateral vessels seen in portal hypertension.

The catheter is now positioned exactly by guiding it with a metal probe under television monitoring to the splenoportal junction. This represents a great improvement in the procedure.

All the catheterizations are performed in the operating theatre, and usually take 20 minutes from the onset of anesthesia. With the catheter maintained at the desired level in the portal vein, the patient is transferred to the department of radiology where the hemodynamic, biochemical and radiological studies are performed.

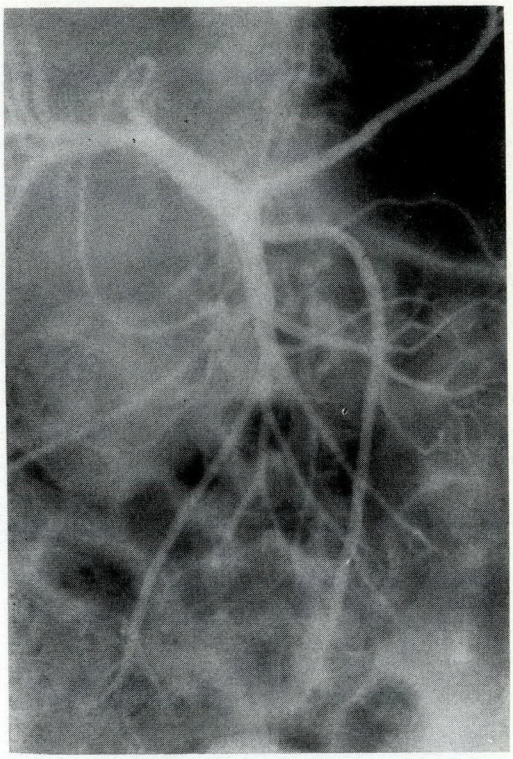

Fig. 1.—Complete retrograde portosplanchnogram made in a patient with cancer of rectum and obtained by injection under high pressure of radio-opaque material. No metastasis in the liver. The end of the catheter is located in the left branch of the portal vein.

RADIOLOGICAL RESULTS

Initially the best we hoped for was that umbilicoportography would be a useful procedure to complement splenoportography. We now believe it is superior to splenoportography, for the following reasons:

(1) It is safer, principally because it avoids puncture of the spleen, which sometimes has serious consquences. In this repect, there is not only the danger of hemorrhage but also the possibility that subsequent splenectomy may be necessary; this must be avoided, especially in children with portal hypertension.

(2) The new technique can be used for patients already splenectomized (post-splenectomy "bleeders"), and for the post-

operative observation of the patency of latero-lateral portacaval shunts.[3]

(3) It allows the precise monitoring of portal pressure under basal conditions and for a definite period of time, a distinct advantage over splenomanometry; biochemical studies can be done, and portography can be repeated at will while the portal catheter is in place.

(4) Better hepatograms and/or portosplanchnograms are obtained, showing most of the tributary veins in their smallest ramifications (Fig. 2).

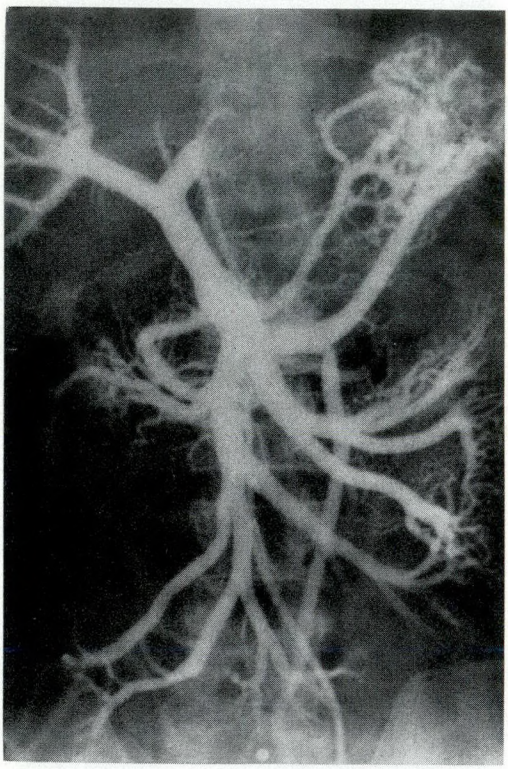

Fig. 2.—Alcoholic hepatitis with a portal pressure of 7 mm.Hg. The catheter has been guided to the level of the splenoportal junction. Complete opacification of the venous splanchnic area has been obtained, as well as of the portal tree, with its tributaries and ramifications in the liver.

When taken with a rapid film changer (Schoenander), the umbilicoportograms are excellent. One of us[5] has classified them under two headings:

(a) Portosplanchnographic, i.e. retrograde opacification of the superior and inferior mesenteric veins, of the splenic vein and of the portal trunk;

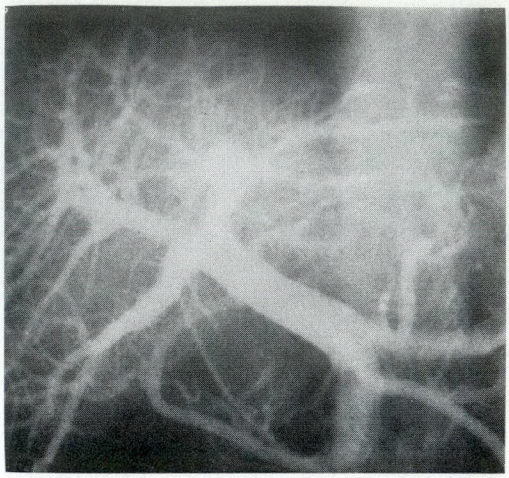

Fig. 3.—Laennec's cirrhosis. Portal hypertension of 16 mm.Hg with evident collateral circulation. Venous phase of hepatogram, three seconds after the beginning of injection.

(b) Hepatographic, divided in three phases:
  i) Venous (Fig. 3)
  ii) Sinusoidal (Fig. 4)
  iii) Parenchymal (Fig. 5).

A somewhat mottled appearance of the hepatogram has also been described in certain cases of cirrhosis (Fig. 6), and has been attributed to a longer sinusoidal phase overlapping the parenchymal one. This appears to be due to an uneven circulation of the dye in the liver, because of different degrees of obstruction.

If splenoportograms can occasionally demonstrate the emergent part of the mes-

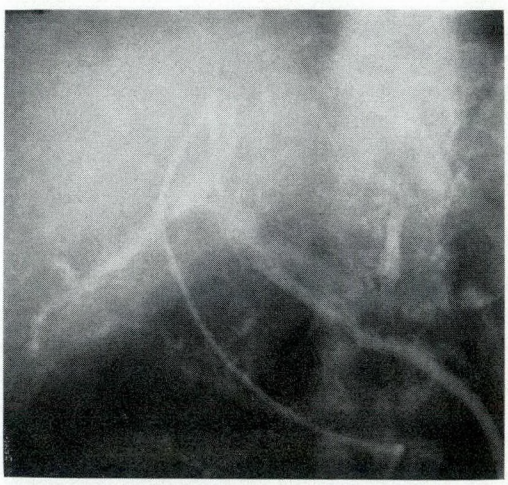

Fig. 4.—Laennec's cirrhosis. Sinusoidal phase of hepatogram, four seconds after injection.

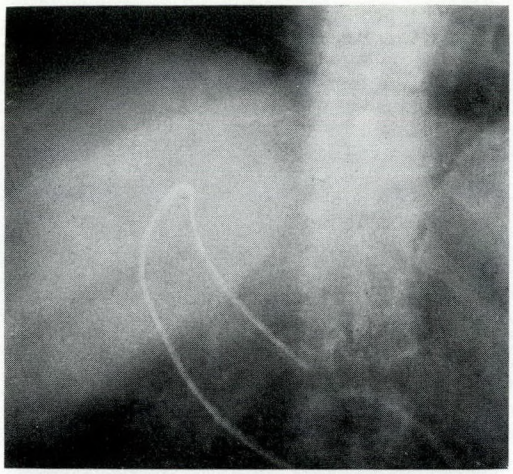

Fig. 5.—Laennec's cirrhosis. Parenchymal phase of hepatogram, seven seconds after injection.

enteric veins, they never permit the detection of abnormal collateral circulation from the mesenteric veins themselves, unless the portal flow has been reversed. Umbilicoportography does (Fig. 7).

The left gastric vein is never opacified during splenoportography under normal conditions,[6] whereas it often shows in portosplanchnograms in patients without significant portal hypertension (Fig. 8).

With the new method, small metastases in the liver have been detected with a high degree of precision. Their exact location was checked afterwards at operation or post mortem (Fig. 9). In two cases, the minimal diameter of a metastasis detected by umbilicohepatography was 2 cm. (Fig. 10). Thus, this technique appears to be at least as accurate as others, i.e. splenoportography, selective hepatic arteriography and liver scanning.[7]

(5) Finally, it is our impression that segmental obstructions of the portal or splenic veins could be identified by this method. This diagnosis could be made by recording

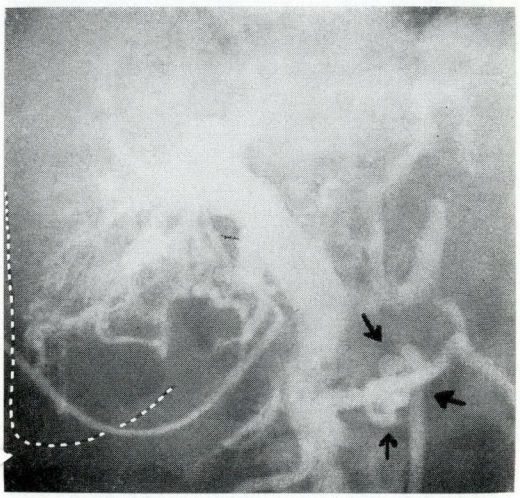

Fig. 7.—Hepatoma superimposed on post-necrotic cirrhosis, with a portal hypertension of 450 mm. of water. Injection of anomalous porto-systemic shunts originating from the mesenteric vein (arrows). Post-mortem studies revealed two neoplastic nodules located at the inferior margin of the right lobe of the liver (outlined on the picture), well shown on the hepatogram.

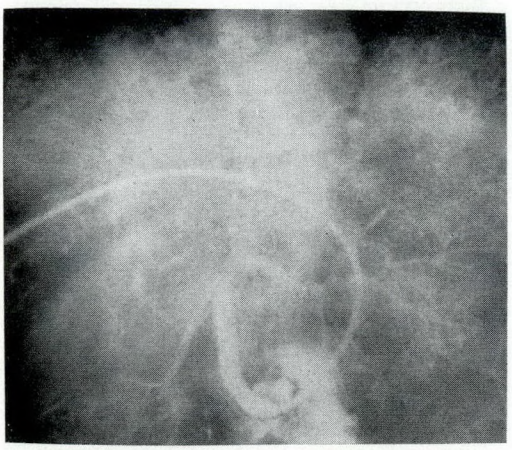

Fig. 6.—Laennec's cirrhosis with portal hypertension of 28.5 mm.Hg. A characteristic heterogeneous pattern of the parenchymal phase, seven seconds after injection, is well shown.

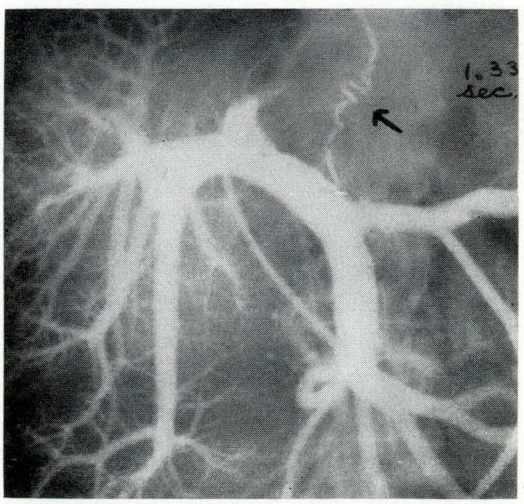

Fig. 8.—Laennec's cirrhosis without significant portal hypertension (13.5 mm.Hg). Opacification of a normal left gastric vein obtained with a catheter placed at the splenoportal junction.

Fig. 9.—Two large metastases of the right lobe of the liver, visualized at the parenchymal phase of hepatogram. The primary tumour was located in the transverse colon.

a normal pressure on the hepatic side of the occlusion in contrast to the elevated pressure that is obtained by splenomanometry while doing splenoportography. In such cases, the latter technique can be considered as an adjunct to umbilicoportography: by the use of both methods, the level and length of the obstruction could be determined.

Hemodynamic and Biochemical Evaluation in Patients with Portal Hypertension

Catheterization of a major right-lobe suprahepatic vein and cannulation of the portal vein allows the determination of the exact level of obstruction in portal hypertension. The precise level of the portal block must be detected in patients with portal hypertension if this pathology is to be understood and adequately cured.

So far, eight patients have been investigated in this manner. All pressures were recorded with the same Statham transducer, positioned 5 cm. below the sternal angle, using the same electronic equipment. The wedged suprahepatic venous pressure and the free portal pressure in cirrhosis, alcoholic hepatitis and hepatic metastases[8] are virtually identical.

In three patients with cirrhosis and two with alcoholic hepatitis, catecholamine levels were sampled in the superior vena

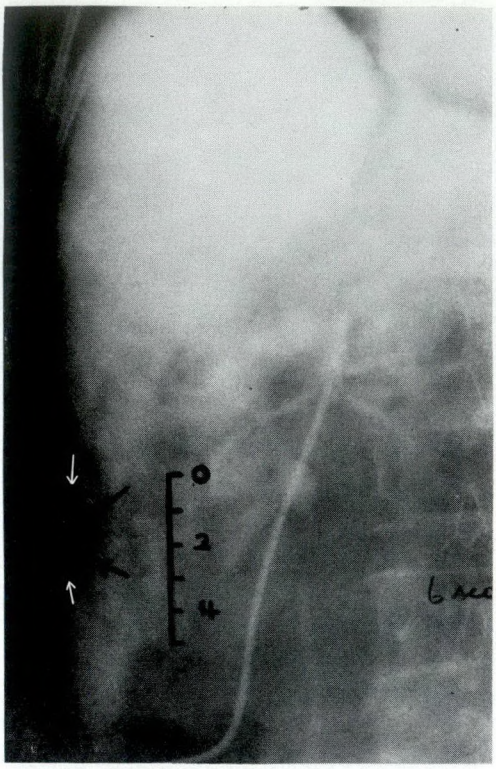

Fig. 10.—A small metastasis, 2 cm. in diameter, in the left lobe of the liver, originated from the rectum. Its exact location was verified during laparotomy.

cava, the inferior vena cava, a right-lobe suprahepatic vein, the portal vein and the right femoral artery. The blood samples were centrifuged and the plasma quick-frozen within 15 min. The catecholamine values were determined using a fluorometric method. No significant differences were found between the value in cirrhotic patients and in those with alcoholic hepatitis, and there was no correlation between the catecholamine level and the portal pressure.[9]

Numerous other applications of this technique are possible, and we feel that it could well become as useful as splenoportography, described by Abeatici and Campi[10] in 1951 and applied soon after to the clinical investigation of portal hypertension by Léger.[11]

Summary

The latest results of portal catheterization *via* the ligamentum teres in 34 patients are presented.

The authors' improvements in this technique are described. These are: better anesthesia with continuous injection of the epidural space; and exact positioning of the portal catheter at the splenoportal junction under radiologic television monitoring to obtain complete opacification of the splanchnic venous system.

The authors believe that umbilicoportography is superior to splenoportography. Characteristic examples of hepatograms and/or portosplanchnograms are shown.

The authors also give preliminary hemodynamic and biochemical results in eight patients with portal hypertension, studied with suprahepatic catheterization of the right lobe of the liver, in combination with portal catheterization *via* the umbilical vein.

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Résumé

Cet article vient compléter un travail sur le même sujet, publié dans ce journal il a un an. Il a pour but de communiquer les derniers résultats du cathétérisme portal par voie ombicale chez trente-quatre malades.

En premier lieu, les auteurs décrivent les améliorations qu'ils ont pu apporter à leur technique. Elles consistent essentiellement en l'obtention d'une anesthésie plus complète au moyen d'une injection continue de l'espace épidural, et en la mise en place au niveau désiré de leur cathéter portal, grâce au contrôle radiologique télévisé, de manière à ce que vraiment toutes les ramifications splanchniques soient opacifiées, et non pas simplement les veines intra-hépatiques, comme il arrivait trop souvent auparavant.

A l'aide d'exemples caractéristiques, on commente ensuite la supériorité possible de l'ombilicoportographie à la spléno-portographie, quant à la valeur des renseignements obtenus, et on en mentionne d'autres avantages.

Les auteurs concluent en donnant les résultats préliminaires, d'un travail en cours, traitant de l'évaluation hémodynamique des blocs portaux, par cathétérisme sus-hépatique combiné au cathétérisme per-ombilical.

ANTHOLOGY OF ORTHOPAEDICS. Mercer Rang. 241 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1966. $7.50.

This interesting book is a true anthology, being a collection of choice passages of literature related to orthopedic surgery. It is a book that will be welcome in the library of orthopedic surgeons and people doing graduate training in this specialty. The author has gone to considerable length to obtain historical information of great interest and biographical data relating to the authors whose writings are quoted. It is particularly enjoyable because of the interesting writings and observations made by men whose names are famous in medical and surgical history. Most of these authors were writing in the nineteenth century, but some were writing well before that time. The book fills a need in a library because it contains much information that is not readily available elsewhere, other than by extensive search of the writings of each author quoted.

The author includes an informative bibliography of classical literature, an index of biographies and a useful page on how to find biographies, portraits and original papers.

Those who read this book will realize, to use the author's own words, "that everything that is old is not old-fashioned" and study of this anthology will help to explain more clearly many of the descriptions, physical signs and methods of treatment that are in common use today.

The book is original in its plan and easily read because of the good writing and careful presentation. It may have special value for teachers as a ready source of information with which they may wish to impress their students.

THROMBOSE PORTALE CHEZ L'ENFANT ET L'ADULTE*

JEAN COUTURE, M.D., F.R.C.S.[C],† *Québec, Qué.*

CETTE affection, qu'on retrouve surtout chez l'enfant, est la cause d'environ 10% des cas d'hypertension portale. Bien qu'il persiste encore certains doutes sur la physiopathologie de cette thrombose, on peut supposer, d'accord avec plusieurs auteurs, qu'il s'agit ici d'une obstruction veineuse consécutive, à l'omphalite du nouveau-né, à une infection généralisée comme la péritonite, à un traumatisme abdominal ou encore à une deshydratation grave.

Durant le stage initial de la maladie, il existe parfois peu de signes cliniques pouvant attirer l'attention de la famille ou du médecin. En effet, la veine porte se recanalise en partie et une circulation collatérale importante se développe dans le hile du foie. L'enfant grandit normalement pendant un certain temps jusqu'à ce que des hémorragies importantes requièrent un traitement énergique.

Selon Arcari et Lynn,[1] 75% des patients ont une première hémorragie avant l'âge de sept ans, et meurent rarement de la première ou de la deuxième hémorragie. D'autre part, ces patients ont un état général très satisfaisant et résistent de façon remarquable aux hémorragies répétées et aux interventions chirurgicales qui leur sont proposées. Il semble que le contrôle des hémorragies, soit par décompression ou ablation des varices, peut procurer à ces enfants une vie relativement normale. Le résultat final ici, est donc beaucoup plus intéressant si on le compare au résultat du traitement des patients atteints d'obstruction intrahépatique, qui finissent toujours par mourir de cirrhose à plus ou moins brève échéance.

Il n'en reste pas moins que le traitement des patients atteints de thrombose de la veine porte avec hypertension portale n'a pas apporté les résultats escomptés. La preuve de cet énoncé se trouve dans le nombre d'opérations qui ont été préconisées dans le traitement de cette maladie (Tableau I).

Comme un grand nombre de ces patients souffrent d'hypersplénisme, on a cru que la splénectomie pourrait corriger l'hypertension portale et empêcher les hémorragies. Nous connaissons aujourd'hui les mauvais résultats apportés par cette opération: en effet, les récidives d'hémorragies sont très frequentes et ces patients présentent alors des problèmes de traitement particulièrement difficiles pour le chirurgien.

L'anastomose spléno-rénale est alors apparue comme un traitement très prometteur: l'enthousiasme fut de courte durée, car on a vite réalisé qu'une telle anastomose avait peu de chances de succès chez un jeune patient en bas de dix ou douze ans et que la thrombose de l'anastomose était fréquente même chez le jeune adulte. L'anastomose porto-cave peut donner de meilleurs résultats. Cependant il est très rare, chez l'enfant, de trouver un segment de veine porte suffisamment développé pour l'exécution d'une anastomose.

Devant l'impossibilité de faire l'une ou l'autre de ces procédures, l'anastomose mésentérico-cave préconisée par Clatworthy et Boles,[2] et utilisée par Voorhees et Blakemore,[3] et Marion, Bouchet et Yon[4] doit être tentée.

Cependant il arrive qu'il soit impossible de faire une anastomose, soit à cause d'un vaisseau de calibre insuffisant ou encore à cause d'hémorragies récidivantes que ne peuvent contrôler les traitements habituels. On peut alors tenter une ligature transésophagienne des varices, procédé qui donne la plupart du temps des résultats

TABLEAU I.— MODES DE TRAITEMENT EMPLOYÉS LE PLUS FRÉQUEMMENT

1—*Interruptions veineuses*
- Splénectomie
- Gastrectomie et bisection

2—*Dérivations veineuses*
- A) *Directes:*
  - Spléno-renale
  - Porto-cave
  - Mésentérico-cave
- B) *Indirectes:*
  - Transposition de la rate
  - Omentacavopexie

3—*Chirurgie directe:*
- Injections sclérosantes
- Ligature transesophagienne
- Esophago-gastrectomie

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†Assistant dans le Service de chirurgie de l'Hôpital du Saint-Sacrement, Québec, Qué.

passagers chez l'enfant mais qui permet parfois de remettre à plus tard l anastomose, alors que le calibre des vaisseaux sera plus adéquat. Selon Britton et Crile,[5] cette intervention n'a donné que 28.6% de récidives chez 28 patients observés depuis 14 ans: cependant beaucoup d'auteurs prétendent que les récidives sont d'au moins 50%.

Comme autre traitement d'attente, il a été suggéré de transporter la rate dans le thorax dans l'espoir de dériver une partie du sang porte. Cette intervention a donné quelques résultats dont la raison d'être est actuellement étudiée dans certains laboratoires.

A la suite de l'insuccès de ces diverses interventions, il arrive parfois qu'on soit obligé, chez certains patients, de faire une chirurgie d'extirpation, soit l'ablation des varices par esophago-gastrectomie. Ce procédé fait habituellement suite à trois ou quatre interventions qui ont créé un climat de frustration tant chez le chirurgien, que chez le patient et sa famille. Quelques points particuliers à cette opération seront discutés en détail un peu plus loin.

Etude clinique

Dans ce travail nous avons étudié 10 patients traités à l'Hôpital du St-Sacrement pour hémorragies importantes dues à des varices ésophagiennes développées à la suite de l'obstruction de la veine porte ou d'une de ses branches. Dans tous les cas, il s'agissait, soit de patients hospitalisés à plusieurs reprises et ayant présenté au moins deux hémorragies graves avant leur admission. Il s'agissait bien d'une obstruction extra-hépatique puisque les tests de la fonction hépatique étaient normaux et que le point d'obstruction était bien mis en évidence par la splénoportographie ou par la laparatomie.

TABLEAU II.—Résultats de la splénectomie. La partie supérieure du tableau nous permet de constater la rapidité d'apparition des hémorragies après splénectomie. A la partie inférieure se trouvent trois patients opérés pour thrombose de la veine splénique

Nom	*Age*	*Opération*	*Résultats*	
J.G.T.	11	Splénectomie	Hémorragies	(2 mois)
R.H.G.	15	Splénectomie et anastomose spléno-rénale	Hémorragies	(1 mois)
G.G.	15	Splénectomie	Hémorragies	(1 mois)
J.P.V.	16	Splénectomie	Hémorragies	(1 mois)
E.G.	37	Splénectomie	Hémorragies	(3 mois)
C.C.	27	Splénectomie	Aucune récidive	(8 ans)
V.G.	42	Splénectomie	Aucune récidive	(8 ans)
C.L.	49	Splénectomie	Hémorragies légères (2)	(7 ans)

La moyenne d'âge des patients était de 22 ans, le plus jeune ayant neuf ans et le plus vieux 49. Pour des raisons évidentes nous avons cru utile d'étudier séparément les manifestations cliniques chez l'enfant et l'adulte. Le pronostic et le traitement diffèrent d'ailleurs dans les deux cas. Les hémorragies de la période infantile sont plus dangereuses et récidivent plus fréquemment, tandis que chez l'adulte les hémorragies deviennent plus espacées, ne se répétant parfois qu'après plusieurs années. Cette constatation nous porte à croire à l'existence de facteurs étiologiques différents chez l'enfant et l'adulte. A la première catégorie appartiennent six patients dont le début des hémorragies a eu lieu à un âge variant de deux à dix ans: chaque enfant a eu au moins une hémorragie sérieuse par année avant de subir un traitement chirurgical. Chez quatre patients d'âge adulte, les hémorragies ont débuté à 27 ans dans un cas et beaucoup plus tard chez les trois autres. Le premier traitement envisagé a été, chez huit patients, une splénectomie seule ou associée à une anastomose spléno-rénale. Dans quatre cas cependant, une tentative d'anastomose avait été infructueuse à cause du petit calibre de la veine splénique.

Le Tableau II met bien en évidence les mauvais résultats de la splénectomie. En effet, chez la majorité de ces huit patients, les récidives hémorragiques sont survenues dans un délai très court, variant de 1 à trois mois, surtout chez les patients plus jeunes. Cependant chez trois patients porteurs d'une thrombose de la veine splénique, le résultat a été excellent.

Nous avons noté de façon particulière, chez nos malades, les constatations hématologiques afin de déterminer l'incidence d'hypersplénisme secondaire. Chez neuf malades, cet état biologique était présent à un stage plus ou moins avancé (Tableau

TABLEAU III.—Hypersplénisme. Noter ici les différences observées dans le volume de la rate chez l'enfant et l'adulte

Nom	*Age*	*Leukocytose*	*Plaquettes*	*Rate palpable*
A.B.	9	N	N	+
C.J.	10	3800	150,000	+
R.H.G.	15	3400	140,000	++
G.G.	15	2800	81,000	++
J.P.V.	16	2300	96,000	++
C.C.	27	5000	100,000	+++
E.G.	37	N	N	+++
V.G.	42	N	90,000	+++
C.L.	49	2300	113,000	+++

III). Nous voyons également que la splénomégalie devient de plus en plus importante si le diagnostic de l'hypertension portale est fait à l'âge adulte. Notons aussi que la splénectomie n'a pas été plus efficace chez un patient atteint ou non d'hypersplénisme, puisque, comme l'indique le tableau précédent, les récidives sont survenues aussi rapidement dans les deux cas.

Tel que dit précédemment, le diagnostic de thrombose portale a été fait dans la majorité des cas par splénoportographie et confirmé par laparatomie et l'enregistrement de la pression portale. Dans l'ensemble, les patients porteurs des plus

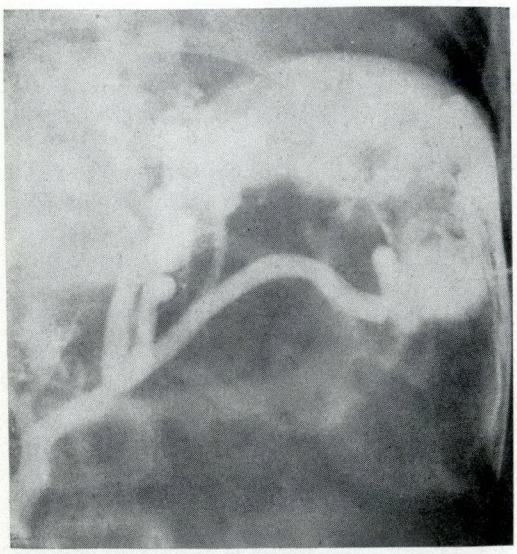

Fig. 1.—A.B., neuf ans, absence de tronc porte: dérivation veineuse impossible.

grosses varices et ayant une pression portale très élevée ont présenté de plus grandes difficultés de traitement. La Fig. 1 montre une splénoportographie représentant un blocage extra-hépatique avec absence d'un tronc porte. Il s'agit d'un enfant de neuf ans chez qui l'anastomose spléno-rénale n'a pas été possible. Dans la Fig. 2, nous voyons une veine splénique plus dilatée et il semble bien qu'il serait possible de faire ici une anastomose spléno-rénale. Cependant l'anastomose a été impossible même après une dissection laborieuse de la veine en tissu pancréatique. Dans la Fig. 3, cependant, le blocage se situe dans le territoire des vaisseaux spléniques: il s'agit ici d'une obstruction de la veine splénique consécutive à une pancréatite aiguë hémorragique traitée six mois auparavant. Cette patiente de 42 ans, a été hospitalisée d'urgence pour hémorragies importantes dues à des varices esophagiennes. Dans ce cas, la splénectomie seule a été le traitement définitif.

Traitements et résultats

Nous avions donc à considérer un traitement chirurgical chez des jeunes patients qui, pour la plupart, avaient été hospitalisés à de multiples reprises pour hémorragies et avaient subi, soit une splénectomie seule ou associée à une anastomose ou une tentative d'anastomose veineuse. Chez ces patients, un traitement définitif s'imposait en raison de la fréquence des hémorragies et d'un danger sérieux pour la vie des patients.

Le Tableau IV nous fait voir le traitement utilisé ainsi que les résultats obtenus à date. La splénectomie a réussi à contrôler presque entièrement les hémorragies chez trois patients: il s'agissait de thrombose de la veine splénique dans les trois cas, tel que démontré par la splénoportographie et la laparatomie.

Chez les sept autres patients, une anastomose mésentérico-cave termino-latérale a été faite dans un cas, tandis que nous avons fait une esophago-gastrectomie chez les six autres. De ce groupe, deux patients ont saigné à nouveau: le premier, un garçon de 17 ans, a fait trois hémorragies postopératoires dont l'importance a diminué avec chaque hospitalisation, en effet, aucune transfusion n'a été nécessaire lors de sa

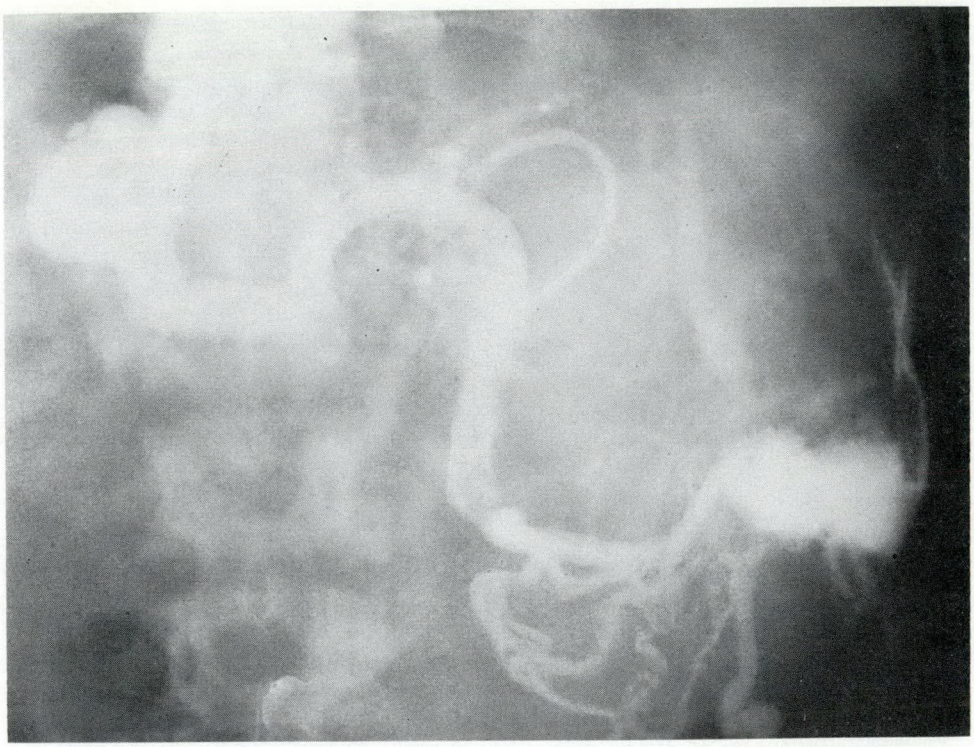

Fig. 2.—J.P.V., 16 ans, thrombose de la veine porte: dérivation veineuse impossible.

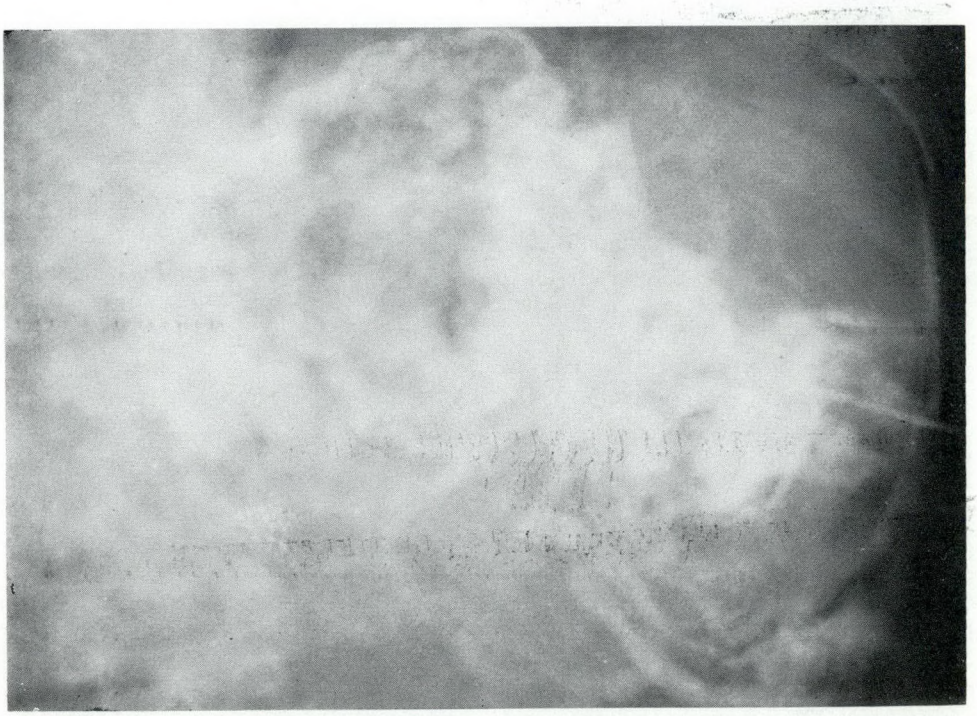

Fig. 3.—V.G., 42 ans, thrombose de la veine splénique après une pancréatite aiguë hémorragique.

TABLEAU IV.—Résultats du traitement chirurgical. Résultats à date chez 10 patients opérés: dans trois cas, apparaissant à la partie inférieure du tableau, le thrombose intéressait la veine splénique. Nous voyons également les résultats chez sept autres patients, dont six ont subi une esophago-gastrectomie, et le septième une anastomose mésenterico-cave. La durée d'observation apparaît entre parenthèses

Nom	*Age*	*Opération*	*Résultats*
A.B.	9	Esophago-gastrectomie	Aucune récidive (1 an)
C.J.	10	Esophago-gastrectomie	Aucune récidive (3 ans)
R.H.G.	15	Esophago-gastrectomie	Aucune récidive (légère anémie ferriprive)
J.P.V.	17	Esophago-gastrectomie	Hémorragies légères (3) (2 ans)
G.G.	19	Anastomose mésenterico-cave	Aucune récidive (5 ans)
J.G.T.	26	Esophago-gastrectomie	Aucune récidive (14 mois)
E.G.	37	Esophago-gastrectomie	Hémorragie-mort (6 mois)
C.C.	27	Splénectomie	Aucune récidive (8 ans)
V.G.	42	Splénectomie	Aucune récidive (1 an)
C.L.	49	Splénectomie	Hémorragies légères (2) (7 ans)

dernière admission. Il persistait chez lui quelques varices résiduelles qui furent traitées avec succès par des injections sclérosantes; incidemment, ce patient avait la plus haute pression portale, soit 48 cm. d'eau. L'autre récidive est survenue chez une patiente de 37 ans qui est morte d'hémorragies six mois après la résection. Cette patiente avait de plus développé une fistule intestinale qui a pu contribuer à la récidive des hémorragies.

En définitive, aucune hémorragie n'est survenue chez sept de nos 10 patients après une période d'observation variant d'un à huit ans. Cependant, selon Léger et Marion[6] on ne peut parler de résultats durables avant un recul d'au moins trois années. Certains de nos malades peuvent donc saigner à nouveau puisque la période d'observation est moins de trois ans chez deux patients et qu'il existe également des possibilités de récidive après trois ans chez les autres.

La technique de résection a été à peu près la même dans tous les cas et a consisté en l'ablation d'au moins la moitié de l'estomac et d'environ deux pouces d'esophage, comme l'ont suggéré Hallenbeck et Adson.[7] Par l'ablation d'une grande partie de l'estomac, nous espérions empêcher la production d'esophagite peptique et également les phénomènes hémorragiques. Un patient seulement a développé une esophagite légère améliorée par un traitement médical; il est à noter qu'une pyloroplastie n'a pu être faite que chez deux patients seulement.

L'opération s'est faite par voie transthoracique chez quatre patients à cause des opérations antérieures qui auraient rendu presque impossible l'opération par voie abdominale. Nous tenons à souligner que ces interventions sont difficiles et compliquées en présence d'hypertension portale et ne se comparent pas à la même opération qu'on ferait dans un cas de cancer.

Ceci explique notre décision de faire une esophagogastrectomie comme premier et seul traitement chez deux jeunes patients de neuf et 10 ans à qui on ne pouvait offrir une dérivation veineuse à cause de leur âge et de l'absence d'une veine splénique de calibre suffisant. Nous avons, chez ces patients, laissé de côté les autres modes de traitement, tels que la ligature transesophagienne des varices, à cause du pourcentage élevé de récidives chez les enfants, tel que mentionné par la plupart des auteurs. L'opération dans les deux cas s'est faite assez facilement par voie abdominale et nous a permis de faire une pyloroplastie. Le résultat est excellent à date et nous espérons avoir évité à ces deux patients les nombreuses hospitalisations rendues nécessaires

TABLEAU V.—Complications de la résection esophago-gastrique. Complications post-opératoires chez les patients ayant subi une résection esophago-gastrique

Rétrécissement anastomotique	3
Abcès sous-phrénique	2
Dehiscence de la plaie	1
Fistule esophagienne	1
Anémie ferriprive	1
Esophagite	1
Total	9 (6 patients)

lorsque le traitement se fait par étapes, comme chez les autres malades.

Ces résultats, cependant, ont été obtenus au prix de nombreuses complications bien qu'aucun malade ne soit décédé dans la période post-opératoire. En effet, neuf complications importantes sont survenues chez six patients (Tableau V); le traitement de ces complications n'a pas présenté en général, de problèmes trop compliqués mais a nécessité plusieurs interventions.

TABLEAU VI.— INTERVENTIONS. NOMBRE D'INTERVENTIONS CHEZ TOUS LES PATIENTS AINSI QUE LE SÉJOUR MOYEN D'HOSPITALISATION: CE NOMBRE COMPREND LES OPÉRATIONS FAITES POUR LE TRAITEMENT DES COMPLICATIONS ET LE TRAITEMENT PAR INJECTIONS SCLÉROSANTES. SI ON EXCLUT CES INTERVENTIONS, ON VOIT QUE 22 OPÉRATIONS ONT ÉTÉ FAITES DANS UN BUT CURATIF

Injections sclérosantes	19
Splénectomie	8
Esophago-gastrectomie	6
Dilatation esophage	6
Drainage abcès	5
Tentative d'anastomose	4
Laparatomie exploratrice	3
Anastomose mésenterico-cave	1
Anastomose spléno-rénale	1
Esophagostomie	1
Total	54 (10 patients)
Séjour moyen	118 jours

Le Tableau VI, illustre bien les différentes phases du traitement chirurgical: 54 interventions ont nécessité un séjour moyen d'hospitalisation de 118 jours par malade.

Ce nombre d'interventions est impressionnant, car il comprend également les opérations faites pour le traitement des complications et le traitement par injections sclérosantes. Si on n'inclut que les interventions faites dans un but curatif, on arrive au nombre de 22, soit 2.2 opérations par patient. A peu près les mêmes constatations ont été faites par Lynn,[8] de la Clinique Mayo, qui rapporte 72 opérations chez 40 patients soit un peu moins de deux opérations par patient.

Pour le chirurgien, le traitement de ces malades demande donc beaucoup de patience et de soins constants pendant plusieurs années. A vrai dire, on ne cesse de voir ces patients, car la guérison, si on peut utiliser ce mot, ne vient qu'après de nombreuses hospitalisations.

DISCUSSION

Malgré les difficultés inhérentes au traitement chirurgical, nous croyons qu'il importe, chez ces jeunes malades, de prendre tous les moyens possibles afin de les protéger des hémorragies répétées et possiblement mortelles. Un traitement agressif par ablation des varices s'impose parfois et doit être fait si, après considération des autres modes de traitement, on croit donner à nos malades des meilleures chances de guérison.

Pour les raisons mentionnées plus haut, nous avons traité six de nos patients par esophago-gastrectomie. Le résultat à date se compare avantageusement aux meilleurs résultats obtenus par dérivation veineuse. Etant donné qu'aucune anastomose n'était possible chez ces patients, on peut dire que l'opération radicale peut être tentée avec de bonnes chances de succès, malgré un taux de morbidité assez élevé. Les complications en général sont survenues chez des patients ayant subi plusieurs opérations antérieures dans le but de corriger les hémorragies.

Cette constatation nous a porté à envisager la résection comme traitement primitif chez deux jeunes patients âgés respectivement de neuf et 10 ans. Encouragés par le résultat obtenu, nous nous demandons si l'esophago-gastrectomie ne devrait pas être employée plus précocement dans le traitement de certains cas de thrombose portale, surtout chez l'enfant.

A l'appui de cette opinion, nous avons signalé le haut pourcentage de récidives hémorragiques rencontrées après les autres traitement chirurgicaux, le grand nombre d'hospitalisations et d'interventions nécessaires chez nos patients, ainsi que les complications importantes rencontrées quand l'esophago-gastrectomie devient le traitement de dernière instance. Le nombre de cas rapportés dans la littérature est cependant trop restreint pour obtenir une appréciation objective des divers traitements.

Nous avons voulu, par cette modeste con-

tribution, exposer le problème sous un aspect nouveau, mais sans prétendre y apporter une solution définitive.

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SUMMARY

Extrahepatic portal hypertension makes up about 10% of all cases of portal hypertension.

Very often shunting procedures are not feasible in many of these patients, either because of a previous splenectomy or because a suitable vein is not available. Radical treatment must then be carried out in order to prevent further episodes of hemorrhage. The surgeon must sometimes undertake an esophagogastrectomy, an operation that carries a high morbidity rate.

The results of treatment in 10 patients operated upon for thrombosis of the portal vein or one of its branches are described. The average age was 22 years; the youngest was nine, the oldest 49. Esophagogastrectomy was done in six; superior mesenteric-caval anastomosis in one; and a splenectomy in three because of thrombosis of the splenic vein. A splenectomy, or a splenorenal shunt had been done previously in all those in whom esophagogastrectomy was done.

Postoperative complications were numerous and important although no patient died in the immediate postoperative period. One patient died from hemorrhage six months postoperatively and two others sustained light hemorrhage after a few weeks.

CONGENITAL CYSTIC ADENOMATOID MALFORMATION OF THE LUNG: A REPORT OF EIGHT CASES*

D. C. BIRDSELL, M.D., P. WENTWORTH, M.D., M.B., Ch.B., B. J. REILLY, M.B., Ch.B. and W. L. DONOHUE, M.A., M.D., F.R.C.P.[C],
Toronto, Ont.

THE uncommon developmental defect, congenital cystic adenomatoid malformation of the lung, is an established entity that has been described in pathological, radiological and surgical literature. It is a form of congenital cystic lung disease. Further communications on this subject are warranted because this lesion can be diagnosed readily and the proper managemnt of it can save the life of an otherwise normal infant. Three children with this lesion were encountered at The Hospital for Sick Children, Toronto in the last year and were successfully treated. A survey of the autopsy and surgical material at this institution back to 1947 provided a further five cases.

The first report of this condition in the English literature was that of Ch'in and Tang[1] from Peking, China in 1949. They collected 10 cases from the German literature going back to Stoerk's report in 1897 and added one of their own. Although the term "adenomatoid" had apparently been used before, they suggested the name "congenital adenomatoid malformation of the lung". A more complete survey of the literature was made by Craig, Kirkpatrick and Neuhauser[2] who added the adjective "cystic". These authors gathered 11 additional cases from the literature and added four of their own. Three of their cases were treated successfully and they cited two previous successes. Recent reports by Herr-

*From the Departments of Pathology and Radiology, The Hospital for Sick Children, Toronto, Ont.
Reprint requests to Dr. W. L. Donohue, Department of Pathology, The Hospital for Sick Children, Toronto, Ont.

mann, Jewett and Galletti,[3] Goodyear and Shillitoe,[4] Spector, Claireaux and Williams,[5] Breckenridge, Rehermann and Gibson[6] and the inclusion of a description of this disease in a recent book,[7] indicate the importance of this malformation in differential diagnosis of respiratory problems in the newborn.

Case Reports

Autopsy Cases

Case 1.—A female infant was admitted to The Hospital for Sick Children, Toronto, on February 28, 1957, 12 hr. after birth because of respiratory distress and cyanosis. The maternal and obstetrical histories were unremarkable. A diagnosis of bilateral atelectasis was made and bronchoscopic suction gave relief, but an area of "consolidation" persisted in the left upper lung field. She remained well for four weeks, but required readmission at five weeks of age because of increasing respiratory difficulty of four days' duration. Examination revealed an acutely ill infant with cyanosis, tachypnea and subcostal indrawing; the heart was shifted to the right. Fluoroscopic examination revealed a left upper lobe density and enlargement of this lobe. Aspiration and atelectasis were diagnosed. Bronchoscopic suction provided temporary relief. However, a febrile course ensued and the infant went on to develop pneumonia and meningitis. Hydrocephalus followed and the infant died at eight weeks of age.

Autopsy revealed that the entire left lung contained cystic areas, many of which contained a purulent exudate. The right lung was collapsed. Examination of the brain revealed a purulent meningitis and obstructive hydrocephalus.

Microscopic sections of the left lung revealed multiple cysts lined by bronchial epithelium and filled with purulent exudate. No cartilage was found in the cyst walls and no direct bronchial communication was seen. The diagnosis was congenital cystic adenomatoid malformation of the lung.

Case 2.—A female infant was admitted to hospital on May 5, 1954, at the age of three days because she vomited all of her feedings. The mother was a healthy, 32-year-old woman, (para ii, gravida ii). The gestation period was 34 weeks and the infant weighed 4 lb. 11 oz. at birth. No respiratory distress was noted. A chest radiograph was reported to be normal. A barium study of the gastrointestinal tract suggested malrotation with obstruction of the small bowel. Laparotomy revealed jejunal atresia, which was treated by resection and primary anastomosis. Four days after operation, the infant developed acute respiratory distress and died after approximately five hours.

Autopsy revealed a left tension pneumothorax with complete collapse of the left lung; microscopically, this lung was atelectatic but otherwise normal. Grossly, the right lung was expanded but, on microscopic examination, areas of atelectasis were seen around multiple cystic spaces lined by bronchial epithelium. These "bronchioles" contained no supporting cartilage.

Case 3.—A male infant was admitted to hospital on July 7, 1957, three hours after birth, with a history of respiratory distress since birth. The maternal and obstetrical histories were unremarkable. Cyanosis and gasping, rapid respirations were noted. The mediastinum was displaced to the right. A chest radiograph was thought to demonstrate a left diaphragmatic hernia; however, this was not confirmed at laparotomy. Bronchoscopy was done immediately. The left main bronchus was too small to be explored; mucus was aspirated from the airway. Air entry was said to be improved but the appearance of the chest film was unchanged. The infant died two hours after operation.

Autopsy revealed enlarged cystic upper and lower lobes of the left lung. The mediastinum was displaced to the right and the right lung was atelectatic. The cyst walls were lined by ciliated columnar epithelium. In places this epithelium projected into the lumen in villous-like folds.

Case 4.—This male infant was born to an 18-year-old primigravida on January 20, 1966, after 35 weeks' gestation. The birth weight was 3 lb. 10¼ oz. At birth the infant was in obvious respiratory distress. A typical Potter's facies was noted, i.e. large, floppy, low-slung ears, deep, long epicanthic folds and an appearance of senility. Also there was an imperforate anus and bilateral talipes calcaneovalgus. At age 10 hr., he was transferred to The Hospital for Sick Children because of bilateral pneumothorax and failure to pass any urine or feces. Laboratory investigations showed severe acidosis. The pneumothoraces were aspirated and intravenous glucose given, but he died 11 hr. after admission, aged 21 hr.

Autopsy revealed bilateral renal agenesis, absent right testicle and imperforate anus. The lungs were hypoplastic; the left weighed 9.8 g. and the right 12.0 g. (normal combined

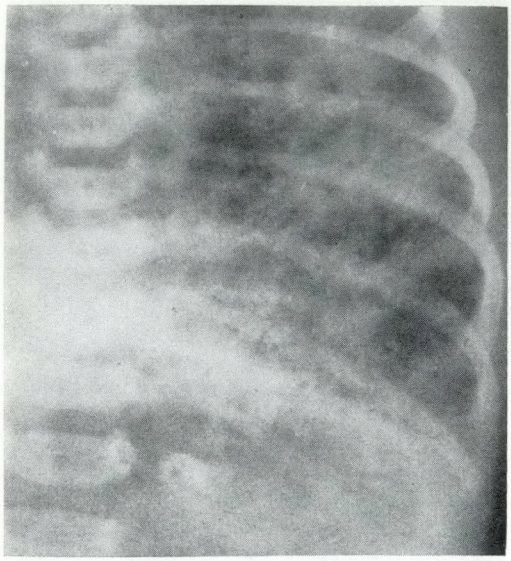

Fig. 1.—Anterior-posterior (AP) view showing cystic adenomatoid malformation of left lower lobe.

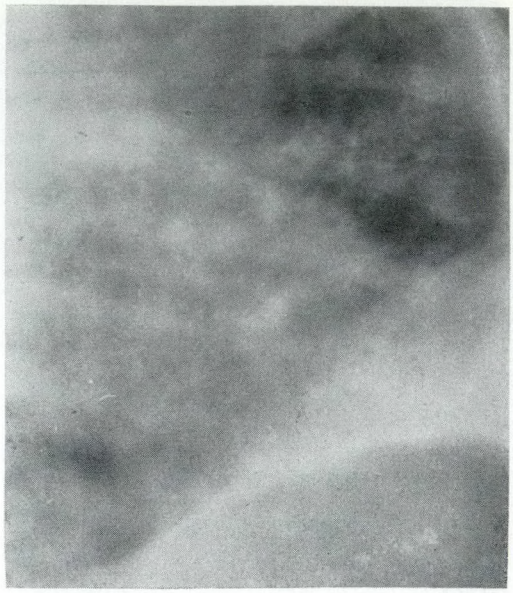

Fig. 2.—Lateral view showing cystic adenomatoid malformation of left lower lobe.

weight is 44.0 g.). The left upper lobe was almost completely replaced by a cystic adenomatoid malformation approximately 2 cm. in diameter.

Surgical Cases

Case 5.—This female infant was born on January 11, 1961, to a healthy, 18-year-old mother (para i, gravida i). Pregnancy, labour and delivery were normal. The infant's birth weight was 6 lb. 12 oz. and her immediate condition was satisfactory. Several hours after birth, cyanosis and respiratory distress developed, and the infant was transferred to The Hospital for Sick Children 14 hr. after birth. The mediastinum was shifted to the right and no breath sounds were heard over the right chest. The radiograph of the chest (Figs. 1 and 2) revealed an enlarged left lower lobe and a diagnosis of congenital cystic adenomatoid malformation was made. Two hours after admission, a thoracotomy was performed and the left lower lobe was removed. Microscopic examination confirmed the diagnosis. The immediate postoperative condition was good. Unfortunately intrapleural drainage failed and the infant developed pneumothorax and died 24 hr. after operation. The autopsy did not reveal any other abnormalities.

Case 6.—This female infant was admitted on February 10, 1965, 26 hr. after birth because of cyanosis and respiratory distress. The maternal and obstetrical histories were unremarkable. The birth weight was 9 lb. 15 oz. Respiratory difficulties and cyanosis were noted within a few minutes. However, the cyanosis was relieved by placing the infant in an oxygen environment. On arrival at The Hospital for Sick Children, respiratory distress and cyanosis were marked. The respiratory rate was 80/min. and indrawing was moderate. The heart rate was 120/min. Physical findings included a shift of the mediastinum to the right, and dullness and decreased air entry over the left lower lung fields. A chest film revealed enlargement and increased density of the left lower lobe; a diagnosis of congenital cystic adenomatoid malformation was made. A barium swallow was performed to rule out a diaphragmatic hernia. Thirty-two hours after birth, a thoracotomy was performed and the cystic left lower lobe was removed. Histological examination confirmed the diagnosis. The postoperative course was not complicated in any way and the infant has remained well.

Case 7.—This boy was first admitted to hospital on August 8, 1960, at the age of 11 months, because of failure to gain weight, a feeding problem and constipation. The mother was a healthy 21-year-old woman and her obstetrical history was unremarkable. At eight months of age the infant was said to have had pneumonia and was treated at home. Examination on arrival revealed a thin, pale, febrile infant with dullness and decreased air entry over the right lung field. A chest radiograph

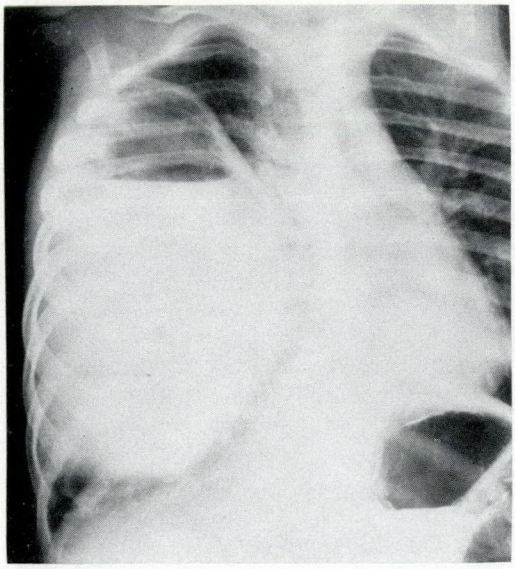

Fig. 3.—Posteroanterior view of chest (upright) showing gas-fluid level in large right lung cyst.

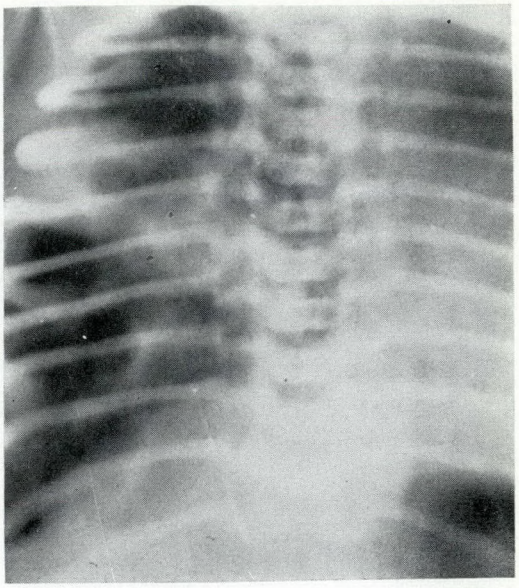

Fig. 4.—AP view showing cystic adenomatoid malformation of right upper and middle lobes.

revealed a mass within the right lung, which was mainly solid but contained some air spaces and displaced the esophagus to the left. A diagnosis of infected bronchogenic cyst was made and a rib resection and drainage were performed. Approximately 350 c.c. of purulent material was drained from an abscess in the right lower lobe. Culture revealed a light growth of *H. influenzae*. A biopsy showed only acute and chronic inflammatory changes in lung tissue.

He was followed as an out-patient and eventually admitted March 2, 1965, for further investigation and treatment. He was small, thin boy who often suffered from respiratory-tract infections. A mild pectus excavatum had developed. Repeated chest films suggested a persistent, infected bronchogenic cyst (Fig. 3). At thoracotomy, the entire right lower lobe was a cystic mass, which on microscopic examination had all the features of congenital cystic adenomatoid malformation. The boy's postoperative course was uncomplicated.

Case 8.—This male infant was admitted on May 25, 1965, at the age of 29 hr., with a history of the sudden onset of respiratory distress at 24 hr. of age. The mother's pregnancy, labour and delivery were normal and the birth weight was 5 lb. 1 oz. His condition at birth was good.

Examination revealed a thin, tense infant in respiratory distress with indrawing and expiratory grunting. Air entry was poor bilaterally. A chest film showed a cystic area of increased density in the right hemithorax and a diagnosis of cystic adenomatoid malformation was made (Fig. 4). At operation, the right upper and middle lobes were found to be involved and were removed. The diagnosis was confirmed histologically. The postoperative course was excellent.

Radiologic Aspects

Where the presenting signs are those of respiratory distress in the newborn, the roentgen findings are sufficiently similar that a precise diagnosis is possible. There is asymmetry of the chest, one hemithorax being larger than the other. The larger half of the chest contains an abnormal lung, which is displacing the heart and mediastinal structures towards the normal side. The pattern within the abnormal lung is that of multiple radiolucent areas varying greatly in size and shape. These cyst-like areas are separated one from the other by strands of dense pulmonary tissue. The precise pattern will depend upon the number, the size and the degree of superimposition of these areas of obstructive emphysema on one another. The involved lung may appear honey-combed or spongy, but occasionally one large cyst may overshadow the others. The condition may be limited to one lobe but, if so, this lobe is usually so large that

positive identification of another normal lobe or lobes is impossible, at least on the basis of a single AP chest film.

The unaffected lung, which is air-containing, is best identified immediately above the leaf of the diaphragm. The upper part of the normal lung is likely to be overshadowed by the heart and mediastinum. The leaves of the diaphragm are, of course, intact.

The spine and bony thorax should be normal. The gas pattern of the bowel seen below the diaphragm is normal.

Differential Diagnosis

Diaphragmatic hernia.—When one hemithorax, usually the left, contains multiple, gas-filled loops of bowel, the abnormal gas pattern seen below the diaphragm is as a rule quite obvious. The leaf of the diaphragm cannot be clearly identified. Before the intrathoracic bowel loops fill completely with gas, the diagnosis may be in doubt, but the introduction of a small amount of either barium or air into the stomach will help to clarify the situation.

Agenesis of one lung.—The complete lack of aerated lung in one hemithorax, the marked herniation of the single lung across the midline and the well-defined, prominent vascular pattern are usually diagnostic. There may be accompanying anomalies of the thoracic spine or ribs.

If, instead of complete agenesis, there is hypoplasia of one lung or atelectasis of an otherwise normal lung, it may mimic cystic adenomatoid malformation but the multicystic pattern is not seen.

Staphylococcal pneumonia.—This condition might be mistaken for a cystic adenomatoid malformation, but only at the stage of multiple pneumatocele formation. Even then, empyema and toxic dilatation of the gut are usually present. Clinical differentiation is straightforward.

Congenital lobar emphysema.—This is another mechanical cause of neonatal respiratory distress. Severe obstructive emphysema of one lobe enlarges the involved hemithorax, compresses the other lobe or lobes of the same lung, and displaces the heart and mediastinal structures towards the normal side. The homogeneously radiolucent pattern of lobar emphysema is usually quite specific.

Neurenteric cyst.—This fluid-filled, posterior mediastinal anomaly is almost always associated with thoracic vertebral anomalies. Even if secondarily infected, it is unlikely to be confused with adenomatoid malformation.

The benign types of congenital cystic disease of the lung in which progressive obstructive emphysema is not a feature are unlikely to be encountered in the neonatal period as a cause of respiratory distress. The cystic emphysema produced by the pulmonary syndrome of Wilson and Mikity[8, 9] is not seen in the neonatal period and, when it eventually develops, is usually bilateral.

When cystic adenomatoid malformation of the lung presents in the manner described in Case 7, the radiographic findings are non-specific and the appearance is that of a large lung abscess. The differential diagnosis would include other gas and fluid-filled cysts of congenital, inflammatory or parasitic nature, or another anomaly such as intralobar pulmonary sequestration with infection and abscess formation.

If an infected cystic adenomatoid malformation lay adjacent to the mediastinum, the differential diagnosis would be that appropriate to a mediastinal mass.[10]

Pathological Aspects

This abnormality shows no tendency to involve one lobe or one lung more than the other. Bilateral involvement has never been reported. The affected lobe or lobes are enlarged and firm. The surface is covered by normal-looking pleura, but the contour reveals underlying cysts of varying size (millimetres to several centimetres). The adjacent normal lung tissue within the same lobe or another lobe is usually displaced and atelectatic. The involved lobe or lobes should be resected.

Grossly, the involved portion of the lung is enlarged and heavy. The external surface has a normal to darkened colour and the appearance described above. The consistency is firm and rubbery. On the cut surface the varying-sized, smooth-walled cysts are seen. Their walls vary in thickness and are homogeneous. Communication with the in-

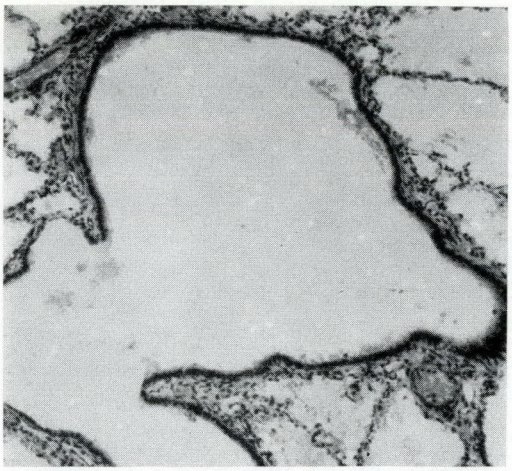

Fig. 5.—Cysts lined by cubo-columnar ciliated epithelium. (H & E)

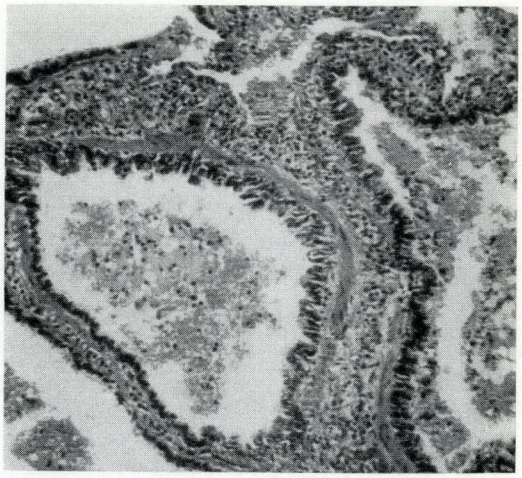

Fig. 6.—A cluster of tubular structures resembling terminal bronchioles. No mucous glands or cartilage are seen but smooth muscle is present. (H & E)

coming major bronchus can be demonstrated.

On microscopic examination, cysts of microscopic size are also seen. The cysts are lined by columnar epithelium, which may be ciliated (Fig. 5). The smaller cysts may be lined by cuboidal epithelium. Scattered clusters of tubular structures are seen resembling terminal bronchioles. Their cells may appear to be secretory in type, but no tubular mucous glands are seen. The walls are made up of compressed alveolar tissue and a fine, loose fibrous stroma. Smooth muscle fibres are occasionally seen in the walls, but no cartilage is found (Fig. 6). The adjacent lung is normal but may show atelectasis from compression.

Discussion

The etiology of cystic adenomatoid malformation of the lung is unknown. It appears to be a true developmental defect and conforms to the definition of a hamartoma, except that one of the tissues indigenous to the lung, namely cartilage, is missing. Other developmental defects that give rise to congenital cysts include accessory lung, sequestrated lobe, bronchogenic cyst and chondromatous hamartoma. These defects probably stem from faults in very early embryologic development, since their cells remain pleuripotential. The fault in congenital cystic adenomatoid malformation occurs later than in congenital cysts, after differentiation to terminal bronchiolar structure has taken place.

No hereditary or maternal factors appear to contribute to this abnormality. The infants described in early reports were mainly prematures with anasarca, and polyhydramnios was often a feature of the pregnancy. However, in recent reports the antecedent pregnancy and the infant have been otherwise normal.

One infant, our Case 4, who had adenomatoid malformation in association with Potter's syndrome seems to be unique. In this case the respiratory difficulty was probably due to pulmonary hypoplasia, the malformation being incidental, although it may have contributed to the pneumothorax.

This anomaly has been considered rare; approximately 35 cases have been reported in the world literature. The fact that four cases were encountered in this hospital during the past year suggests that it may not be extremely uncommon after all. An increased awareness of the anomaly probably accounts for the increasing number of case reports.

The condition usually presents in the first few hours or days of life as respiratory distress, which is related to the increased size of the affected lung. The condition may present later in childhood, as in our Case 8. In these late cases, secondary infection is the mode of presentation.[3]

The onset of symptoms and the progressive nature of the condition are related to

the increasing size of the cystic lung as air becomes trapped within it. Bronchial communication allows air to enter, but because there is no cartilage in the walls of the cysts, the openings collapse on expiration.

The neonate shows signs of respiratory distress with cyanosis, tachypnea and indrawing. The other signs are related to the site and size of the enlarged cystic lung. The affected region will be dull to percussion and air entry will be poor. The mediastinum may be shifted to the unaffected side.

Usually the infant is otherwise normal. No associated anomalies have been found, except in our Case 4 in which this condition occurred with Potter's syndrome.

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Résumé

Il ressort d'une revue de la littérature sur les malformations adénomatoïdes kystiques congénitales du poumon qu'il s'agit d'une maladie kystique du poumon peu fréquente, mais d'un type bien connu. On en a rapporté huit cas observés à The Hospital for Sick Children de Toronto. Deux cas récents ont été diagnostiqués dans les quelques heures qui ont suivi la naissance et le traitement chirurgical rapide a réussi. Un troisième cas a été découvert chez un enfant qui présentait un abcès pulmonaire chronique.

Les signes qu'on observe chez un nouveau-né sont la détresse respiratoire et une masse intrapulmonaire qui déplace les structures normales. La radiologie peut avoir une valeur diagnostique. La résection chirurgicale du lobe ou des lobes malades se traduit par la guérison. Le nourrisson est par ailleurs normal et le pronostic final est excellent.

THE DIFFERENTIAL DIAGNOSIS OF HEMOBILIA

The typical symptoms of hemobilia are pain, jaundice, hematemesis, and melena. The pain is of the biliary colic type. Obstructive jaundice occurs because blood and clots prevent the passage of the bile to the intestine. Massive hematemesis or melena may lead to shock and, subsequently, secondary anemia. If the bleeding is into the gallbladder or if clots prevent emptying of the gallbladder, the organ may be palpated below the right costal margin . . .

Severe inflammation around the biliary tract may predispose to formation of an aneurysm in an arteriosclerotic vessel. Trauma to an artery during operation also may occur in inflamed tissue. Pressure necrosis and choledochoarterial fistula may be caused by a T-tube. . . . When the hepatic artery is involved there is the danger of hepatic necrosis, which requires large doses of antibiotic drugs along with supportive therapy, and when necessary, one should be prepared to carry out partial hepatectomy . . .

Since the source of acute massive hematemesis and melena eludes specific diagnosis in 5 to 15% of cases, hemobilia should be considered a possible cause. If hemobilia is found, operative cholangiography is indicated and, in the absence of an intraductal lesion, one must be prepared to proceed with a portography and hepatic arteriography to rule out an intrahepatic aneurysm or tumour as a source of the bleeding.—Larmi, T. K. I.: Hemobilia associated with cholecystitis, postcholecystectomy conditions and trauma: review of 12 cases, *Ann. Surg.*, **163:** 379, 1966.

FREE PERFORATION IN MALIGNANCIES OF THE STOMACH

T. S. WILSON, M.D., F.R.C.S.[C], F.A.C.S.,* *Edmonton, Alta.*

In the past 10 years (1956-1965), I have had two patients with free perforation of carcinoma of the stomach. Both these patients presented in the same way—with dramatic suddenness, and symptoms and signs of acute perforation of a hollow viscus. Both cases were misdiagnosed preoperatively as perforated peptic ulcer—probably duodenal. At operation the findings were identical: in each patient, a large ulcer on the anterior wall of the antrum of the stomach had a free perforation in its centre and there was gross peritoneal soiling with gastric contents. Both patients were thought to have benign gastric ulcers at operation, both were treated by an immediate gastrectomy and both had a smooth postoperative course.

The first patient died in his home town four months after operation. Autopsy was not carried out but presumably he died from carcinomatosis. The second patient was operated on in February 1965, and in the short time since operation has been well. However, on follow-up examination on May 12, 1966, there was a hard nodule in the umbilicus, which was probably secondary carcinoma.

This experience stimulated me to review the literature on the subject and the total experience in the hospitals in this area.

Historical Background

On May 5, 1821, Napoleon I died on St. Helena Island at the age of 52 years. There is a difference of opinion about the cause of death but Bechet[1] claims an autopsy showed a carcinoma of the stomach with perforation. Another facet of Napoleon's genius is reflected in his final instructions. "After my death I wish you to make an autopsy. . . . Examine well my stomach, and make a detailed report to my son. Indicate to him what remedies or mode of life he can pursue which will prevent his suffering from a similar disease. This is very important for my father died of a cirrhosis of the pylorus with symptoms very much like mine." Laennec[2] in 1824, is given credit for the first pathological description of perforation of a gastric carcinoma.

Aird[3] in 1935, collected 79 patients with perforation of gastric carcinoma but only 44 of these showed free perforation into the general peritoneal cavity. He distinguished between the fulminating or classical perforation, and the silent or localized type. The former made up two-thirds of the total. He suggested that immediate gastrectomy was the treatment of choice.

McNealy and Hedin[4] reviewed 133 patients seen between 1925 and 1937. No part of the stomach was immune from perforation, but most occurred in the prepyloric area and the lesser curve. Of 63 patients operated on, 37 died—an immediate hospital mortality of 59%—and 13 more died within two months. The 13 survivors were not followed: five had resection, four gastroenterostomy, three simple closure and one gastrostomy. In the opinion of these authors, the condition was so desperate that the best operation was closure of the perforation, with or without a gastroenterostomy. If the patient survived, this procedure could be followed at a later date by resection.

Bisgard[5] collected 217 cases with perforation of gastric malignancy from the literature up to 1945. He believed that many more such patients either were not reported or died undiagnosed. In only seven of the 217 patients was the correct diagnosis made before operation or post mortem. In many, the correct diagnosis was not made at operation. Simple closure of the perforation was most commonly carried out, but this was followed by 80% mortality; following gastric resection, done in 15 cases, there were only two deaths—a mortality rate of 13%. However, Bisgard emphasized that gastrectomy was probably done in the more favourable patients.

Guiss,[6] in a collective review of 2891 patients with cancer of the stomach, found 108 patients (3.7%) with acute perforation. Thus it was an uncommon incident in the natural history of the disease.

Heimlich[7] reported a patient who sur-

*Department of Surgery, University of Alberta and University of Alberta Hospital, Edmonton, Alta.

vived for seven years following immediate gastric resection, but finally died of carcinomatosis peritonei. Emmett,[8] who resects perforated ulcers, had a patient who lived eight years after gastrectomy for a perforated gastric carcinoma. This was the longest survivor I could find. He finally died of a massive recurrence.

Local Experience

Most patients with carcinoma of the stomach in northern Alberta pass through the Edmonton Branch of the Cancer Diagnostic Clinic, Department of Public Health of Alberta. This serves about 700,000 people. During the 13-year period 1953-1965, 1079 new patients have been seen with malignancy of the stomach. Of these, 14 perforated—an incidence of 1.3%. Undoubtedly some perforations were missed, particularly in terminal cases that did not come to post mortem, so the incidence is probably higher. A short case history of each of these 14 patients (Table I) follows:

TABLE I.—Cases of Perforated Gastric Malignancy

	Name	Sex	Age	Date of perforation	Preoperative diagnosis	Pathological diagnosis	Treatment	Early course	Death (months)
1.	S.T.	M	54	Aug. 21, 1954	Perforated duodenal ulcer	Carcinoma stomach	Patch	Good	5
2.	E.K.	M	25	May 29, 1955	Perforated duodenal ulcer	Lymphosarcoma stomach	Gastrectomy	Good	5
3.	H.S.	F	71	May 5, 1956	Terminal carcinoma	Carcinoma stomach	Nil	Death	0
4.	J.R.	M	52	Aug. 15, 1956	Perforated gastric ulcer	Carcinoma stomach	Gastrectomy	Good	4
5.	G.C.	M	58	Jan. 16, 1957	Perforated duodenal ulcer	Leiomyosarcoma stomach	Gastrectomy	Good	15
6.	C.T.	M	70	July 20, 1957	Terminal carcinoma	Carcinoma stomach	Nil	Death	0
7.	O.L.	M	69	Jan. 3, 1958	Perforated duodenal ulcer	Carcinoma stomach	Patch, later gastrectomy	Good	41
8.	M.S.	F	52	Oct. 22, 1960	Terminal carcinoma	Carcinoma stomach	Laparotomy	Death	0
9.	A.S.	M	79	May 7, 1962	Perforated duodenal ulcer	Carcinoma stomach	Drainage, later gastroenterostomy	Good	10
10.	J.C.	M	69	Aug. 18, 1962	Perforated gastric ulcer	Carcinoma stomach	Gastrectomy	Good	7
11.	F.A.	M	60	Mar. 3, 1964	Perforated duodenal ulcer	Carcinoma stomach	Patch, later gastrectomy	Good	well
12.	A.L.	M	80	Mar. 9, 1964	Perforated duodenal ulcer	Carcinoma stomach	Patch + gastrostomy, later gastrectomy	Good	9
13.	A.N.	M	45	Feb. 17, 1965	Perforated gastric ulcer	Carcinoma stomach	Gastrectomy	Good	Metastasis navel
14.	R.T.	F	77	June 2, 1965	Terminal carcinoma	Carcinoma stomach	Gastroenterostomy before perforation	Death	0

Case Reports

Case 1.—S.T., a 54-year-old man, had indigestion in May 1954 and a gastric ulcer was demonstrated on barium meal. Suddenly, on August 22, 1954, he developed acute epigastric pain with radiation to the right shoulder. The abdomen was tender and rigid. On plain upright film, air was seen under the diaphragm.

A diagnosis of perforated peptic ulcer was made and a laparotomy was done four hours after the onset. A considerable volume of gastric contents was found in the peritoneal cavity and there was an early peritonitis. An umbilicated nodule was seen in the right lobe of the liver. In the prepyloric region of the stomach, there was an indurated mass and a free perforation, 0.2 cm. in diameter, was seen near the lesser curve. An omental patch was used to close it. Biopsy of the nodule in the liver showed metastatic carcinoma.

He did reasonably well and a "second-look" laparotomy was done on September 8, 1954. The lesion in the stomach was considered to be inoperable; a biopsy of a lymph node showed metastatic carcinoma. He was discharged from hospital. However, by November 24, 1954, his condition was deteriorating. He had considerable upper abdominal pain and was vomiting.

He died on January 26, 1955, five months after the perforation.

Case 2.—E.K., a 25-year-old man, had stomach trouble for six months and marked epigastric pain for three days. At 7:20 p.m. on May 29, 1955, he developed sudden, severe, upper abdominal pain and, when seen two hours later, was thought to have a perforated peptic ulcer. Immediate laparotomy was done. A considerable amount of gastric contents was found in the peritoneal cavity and a mass was seen on the mid-portion of the anterior wall of the stomach with a "large" perforation in the centre.

An immediate Billroth II gastrectomy was carried out. His postoperative course was uneventful and he went home.

Pathological examination revealed a lymphosarcoma of the stomach. The lymph nodes were negative.

He was given a course of Co^{60} in July 1955.

On September 12, 1955, there was a suggestion of a mass in the epigastrium. He had occasional vomiting. By October 4, 1955, a definite large mass could be felt. A radiograph of his chest was negative.

A second course of Co^{60} was given but, by October 27, 1955, definite ascites was demonstrated and his course was progressively downhill.

He died on November 14, 1955, 5½ months after the perforation.

Case 3.—H.S., a 71-year-old woman, had a laparotomy on April 9, 1956, which showed a large mass in the pyloric end of the stomach, and massive secondaries in the liver. A gastrojejunostomy was carried out. She did poorly postoperatively and died on May 8, 1956.

Autopsy revealed perforation of a gastric carcinoma with generalized peritonitis and carcinomatosis peritonei. There was secondary metastatic cancer in her liver and lungs.

In this patient the perforation was a terminal event.

Case 4.—J.R., a 52-year-old man, had ulcer symptoms beginning in 1950, which became more severe in December 1955. In January 1956, a barium meal showed a small gastric ulcer on the lesser curve of the stomach in the antrum. It was thought to be benign.

About August 1, 1956, epigastric pain developed, which radiated to the back and was only partially relieved by soda, milk and food. He began to vomit old blood and passed tarry stools about August 10, 1956. He had lost 30 lb. over the previous six months.

On August 14, 1956, a second barium meal was done. At 3:00 a.m. on August 15, 1956, on the train coming into Edmonton (200 miles), sudden, severe epigastric pain developed. In the emergency department 10 hr. later he was in moderate shock and had a pulse rate of 110/min. The abdomen was generally tender and rigid, and it was considered that he had a generalized peritonitis due to a perforated peptic ulcer. Liver dullness was diminished and, on rectal examination, pelvic tenderness was demonstrated. His leukocyte count was 13,000/c.mm. with 72% polymorphs.

Laparotomy was done 13 hr. after the onset on August 15, 1956. The peritoneal cavity was filled with gastric contents and barium. He had an early generalized peritonitis, subsequently found to be due to *E. coli*. There was a large anterior-wall gastric ulcer on the lesser curve at the angle, surrounded by a 5-cm. area of local induration; there was a ragged perforation in its centre, 1 cm. in diameter. I did not believe a patch would hold and because he had also been bleeding for five days a 50% Billroth II gastrectomy was carried out, despite the peritonitis and the presence of considerable barium in his abdomen.

His postoperative course was smooth and by August 18, 1956, he was taking fluids by mouth. He was discharged well on September 3, 1956, on the nineteenth postoperative day. The incision healed by first intention.

He died in the country on September 21, 1956, four months after the perforation. No post mortem was done.

Case 5.—J.C., a 58-year-old man, had some indigestion for one year, but 24 hr. before operation (January 16, 1957), he developed sudden, severe abdominal pain. The presence of general tenderness and rigidity led to a preoperative diagnosis of perforated peptic ulcer.

At laparotomy, he had an orange-sized mass in the mid-portion of the stomach arising from the greater curve; there was a perforation, 0.5 cm. in diameter, on the anterior surface. There was a single metastasis in the left lobe of the liver. A Billroth I gastrectomy was carried out. Convalescence was smooth. He left hospital in two weeks.

He died on April 21, 1958, one year and three months after his operation.

The pathological report on the resected portion of the stomach and tumour was interesting. "The specimen consisted of a small portion of stomach with a firm mass, grey-brown in colour, and 8 cm. in diameter on the greater curve. There was a perforation 0.5 cm. in its centre. Sections showed a rapidly growing *leiomyosarcoma of the stomach* with spread to omentum."

Case 6.—C.T., a 70-year-old man, had a laparotomy for a supposed intestinal obstruction on June 5, 1957. Carcinomatosis peritonei was found and a large tumour in the cecum, which was thought to be the primary. Ileotransverse colostomy was done. Biopsy of the omentum showed carcinoma. He returned home for a short time after a normal convalescence.

On July 21, 1957, he was readmitted with a 24-hr. history of vomiting and abdominal distension. There was a rapid downhill course and he died 18 hr. after admission.

At post mortem, he had acute peritonitis. There were 500 c.c. of yellow turbid odorous fluid in the abdomen and numerous fibrinous tags. The peritonitis was due to a perforation in the anterior wall of the stomach near the pylorus measuring 0.2 cm. in diameter, in an

ulcer measuring 2.5 cm. There were matted nodes along the lesser curve and also gross involvement of the omentum. The cecal mass was secondary tumour. On microscopic section he had adenocarcinoma of the stomach. The perforation was a terminal event in this illness.

Case 7.—O.L., a 69-year-old man, had no previous history of ulcer but a recent weight loss of 10 lb. Suddenly at 4:30 a.m. on January 3, 1958, he was seized with sudden, severe epigastric and substernal pain radiating to the left shoulder. It gradually spread over his abdomen. He was moderately dyspneic and was afraid to move. He had fainted at home. Examination in hospital showed abdominal tenderness and board-like rigidity.

A diagnosis of perforated peptic ulcer was made and operation carried out four hours after the onset. A small perforation—anterior wall, pyloric antrum—was found, with gastric contents in the abdomen and an early generalized peritonitis. An omental patch closed the hole satisfactorily.

Convalescence was uneventful and after he recovered, on February 5, 1958, a Billroth II gastrectomy was carried out. The previous patch had held, but there was a mass in the pyloric antrum. Sections from the specimen showed an anaplastic, infiltrating adenocarcinoma but the lymph nodes were negative. Again his convalescence was uneventful and he was discharged on February 21, 1958.

He enjoyed two years of fairly good health and then developed signs of large-bowel obstruction. On February 1, 1960, a sigmoid colostomy was carried out because of metastatic carcinoma in the sigmoid and upper rectum from the primary in the stomach.

Palliation with Co^{60}, Cs^{137} and cyclophosphamide was attempted and by December 15, 1960, he was still remarkably well.

He died from generalized carcinoma on June 25, 1961, three years and five months after the perforation.

Case 8.—M.S., a 52-year-old woman, had been in ill health for one year, with loss of appetite and a weight loss of 15 lb. Three days before admission she developed epigastric pain, which became very severe on October 22, 1960, the night before admission.

On admission (October 23, 1960), she was acutely ill, had a pulse rate of 120/min. and was dehydrated. The abdomen was distended, tympanitic with generalized tenderness, rigidity and no bowel sounds. A plain film failed to show free air. On rectal examination there were pelvic implants—a "rectal shelf".

The same day, laparotomy was carried out in desperation. She had 4 to 5 l. of fluid in the abdomen, generalized peritonitis and carcinomatosis. She died the following day.

At post mortem there was a large lesser curve ulcer, 5 cm. in diameter, on the anterior wall of the pyloric antrum, with a perforation in its centre. There was generalized carcinomatosis peritonei. Microscopic section showed a rapidly growing adenocarcinoma of the stomach.

This perforation was a terminal event in a patient with carcinomatosis. It may have occurred the day before operation.

Case 9.—A.S., a 78-year-old man, was admitted to hospital on May 14, 1962, with epigastric pain and vomiting of one week's duration. He was ill, had a temperature of 101°F. and upper abdominal tenderness. A barium meal showed a perforation of the lesser curve of the stomach, with a sinus tract extending to the right to outline a large subphrenic abscess (Fig. 1).

On May 16, 1962, the abscess was drained through a right subcostal incision and 1500 c.c. of purulent material was obtained, which on culture grew *Staph. aureus*, *E. coli*, Proteus, and *Strept. fecalis*. At this time the abscess was thought to be due to a perforated duodenal ulcer. Drainage was persistent but gradually subsided, and following a transurethral resection on June 4, 1962, he was discharged walking on July 6, 1962.

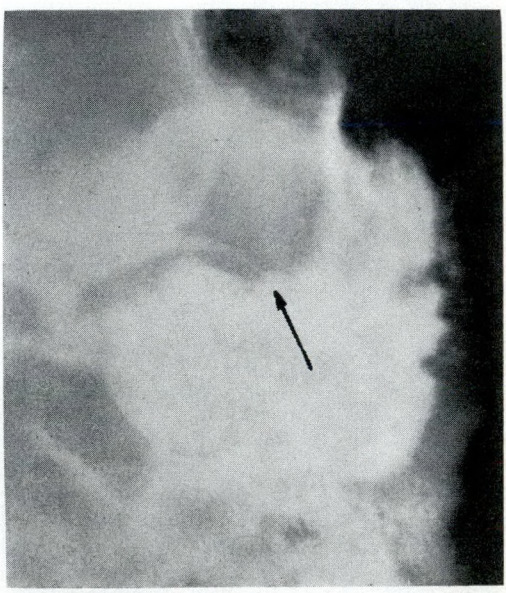

Fig. 1.—Barium meal showing leak from lesser curve of stomach into a large subphrenic abscess cavity. Arrow marks site of perforation (Case 9).

He was readmitted October 18, 1962, complaining of weakness. He had a distended abdomen due to ascites, and a large mass in the epigastrium. His hemoglobin was 4.5 g.%. Following blood transfusions on October 13, 1962, biopsy of a prepyloric mass and gastrojejunostomy was done. He had adenocarcinoma of the stomach—mucoid type.

He died on March 12, 1963, 10 months after the perforation.

Case 10.—J.C., a 69-year-old man, had a 13-year history of "duodenal ulcer". In May 1962, he had a barium meal that showed a gastric ulcer in the antrum probably benign, but malignancy was suggested. Because he had had a second coronary thrombosis in January 1962, he was treated medically.

He was admitted to hospital on August 18, 1962, with a four-hour history of severe pain that had begun suddenly in the upper abdomen and then spread all over. He was in shock, had a board-like abdomen and no bowel sounds. A plain upright film showed a considerable quantity of free air under both diaphragms.

At exploration (August 18, 1962), on opening the abdomen, a large amount of gas and stomach contents were noted and there was an early generalized peritonitis. A large lesser curve ulcer on the anterior wall of the pyloric antrum was seen. The ulcer measured 4 cm. in diameter, and there was a 0.2 cm. perforation in its centre. Palpable nodes were found near the mass. Because the lesion could have been either benign or malignant, a Billroth II gastrectomy was carried out.

On section, an adenocarcinoma of gelatinous type was recognized. Four lymph nodes were found and examined and all showed metastatic carcinoma.

He made a good recovery except for a Proteus wound infection and was discharged on September 20, 1962. He was fairly well until February 1963 when ascites developed. A course of 5-fluorouracil was administered without effect.

He died on March 28, 1963, seven months after the perforation.

Case 11.—F.A., a 60-year-old man, was believed to have a gastric ulcer in 1962, on the basis of radiological examination. He was treated medically but the ulcer did not heal completely.

On March 3, 1964, he developed sudden, severe epigastric pain. The abdomen was rigid. A small amount of free air was seen on a flat plate of the abdomen. The preoperative diagnosis was perforated peptic ulcer and, at operation, an ulcer was found on the lesser curve at the angle. It had perforated anteriorly. The possibility that the ulcer was malignant was considered. An omental patch was applied. Postoperatively he developed left lower lobe atelectasis but no abdominal complications. He was discharged in three weeks.

Gastrectomy was done on June 2, 1964. At this time a large mass was found in the lesser curve extending posteriorly to the pancreas. A Billroth I (75%) gastrectomy was carried out. Grossly local nodes appeared to be involved. A biopsy of the pancreatic capsule was taken.

Postoperatively he did well except for a *Staph. aureus* wound infection. He left the hospital in 19 days. At the last follow-up in February, 1966, he was feeling well and gaining weight. A barium meal showed no evidence of recurrence. There was no clinical evidence of metastases.

On pathological examination of the gastrectomy specimen, a large ulcerating carcinoma 3.5 cm. in diameter was noted, which had firm rolled edges and a necrotic base. The tumour extended through the wall of the stomach to involve the serosal surface. Histologically, the tumour was an anaplastic adenocarcinoma. The biopsy from the pancreatic capsule also showed anaplastic carcinoma.

Case 12.—A.L., an 80-year-old man, had a sudden attack of severe upper abdominal pain about March 9, 1964, and was treated conservatively in his local hospital. Over the next week, a tender mass developed in the left upper quadrant of the abdomen with some rebound tenderness. Following admission to hospital on March 15, 1964, plain films of the abdomen showed air under the right diaphragm. A diagnosis was made of perforated duodenal ulcer with abscess formation. At laparotomy on March 17, 1964, a perforation of a gastric ulcer was found on the anterior wall of the lesser curve near the angle, and a localized abscess.

The hole was closed with an omental patch. A gastrostomy was done and a Foley catheter was left in place. Culture from the abscess yielded *Cl. welchii (perfringens).*

His postoperative course was fairly smooth. Gastric washings were negative for malignant cells. On April 16, 1964, the ulcer was inspected with a cystoscope through the gastrostomy opening. A good view of the ulcer was obtained and it was thought to be benign.

He was discharged on April 23, 1964, to a convalescent hospital. Moderate bleeding developed about June 1, 1964. On barium ex-

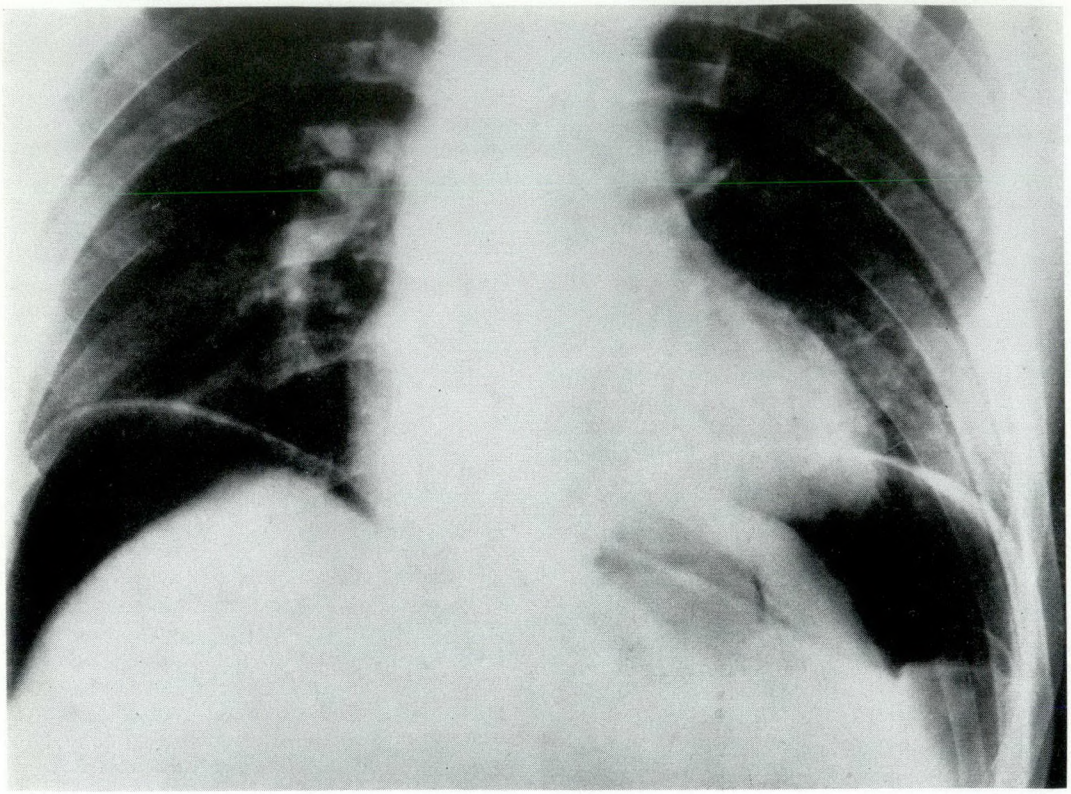

Fig. 2.—Erect plain film of abdomen showing large amount of air under both diaphragms (Case 13).

amination a probable carcinoma of the stomach was seen. A Billroth II gastrectomy was carried out on June 10, 1964. Again, considering his age, his postoperative course was fairly smooth. He was discharged to a nursing home on June 23, 1964, where he died on December 1, 1964, nine months after his perforation.

Histologically the gastrectomy specimen showed an anaplastic carcinoma.

Case 13.—A.N., a 45-year-old man, was first seen in February 1964, with a one-month's history of epigastric pain relieved by food and milk. A barium meal showed a gastric ulcer in the prepyloric area, thought to be benign. Four weeks later, after medical treatment with rest, diet and alkalis, a second barium meal showed considerable healing of the ulcer. He was well until two weeks before admission to hospital when he developed recurrent epigastric distress relieved by soda. However, he kept working as a lumberjack, and, while cutting down a tree at about 11:30 a.m. on February 17, 1965, was seized with a sudden severe upper abdominal pain that was "like being hit by an axe". During the 250-mile trip to Edmonton, in the back of a half-ton truck, he fainted twice.

Eight hours later when seen in the emergency department, he was in moderate shock with a rigid, tender abdomen, absent bowel sounds and pain on rectal examination. A plain upright film of the abdomen showed considerable air under both leaves of the diaphragm (Fig. 2). Laparotomy was carried out two hours after admission and 300 c.c. of purulent material in the abdomen were removed by suction. Afterwards *Strep. viridans* was isolated. He had a large prepyloric ulcer, anterior wall, nearer the lesser curve, which measured 3 cm. in diameter and had a perforation in the centre 0.5 cm. in diameter. This was thought to be benign, and because of his precarious condition, an attempt was made to patch the hole with omentum but the sutures just pulled out. A 50% Billroth I gastrectomy was then carried out.

Convalescence was uneventful, he was eating solid foods in one week and was discharged on the eleventh postoperative day.

Eight days later he was readmitted for five days with a wound infection, which drained spontaneously. Anaerobic streptococci were cultured from the discharge.

By May 1, 1965, he was feeling very well, eating normally, and had gained 10 lb.

He was well in January 1966. Radiographic examination of his gastric stump was normal. However on May 12, 1966, a hard nodule was noted in his navel—probably secondary carcinoma.

The resected specimen showed a deep ulcer in the prepyloric region, 2 cm. in diameter, with a ragged perforation. The lesion was an ulcerating adenocarcinoma; two nodes of three on the lesser curve and one node of three on the greater curve showed carcinoma.

Case 14.—R.T., a 77-year-old woman, was admitted on May 16, 1965, with a history of weight loss and anorexia of one year's duration. There was a suggestion of an epigastric mass. On May 31, at laparotomy, an inoperable carcinoma was found arising in the gastric antrum. A gastrojejunostomy was done.

Postoperatively the patient developed a fever of 101°F. and abdominal distension with signs of peritonitis. She died on June 5, 1965.

At post mortem, a spontaneous perforation of a carcinoma in the antrum of the stomach was found, with generalized peritonitis. The perforation was on the anterior wall. Two litres of cloudy brown fluid was aspirated from the abdomen—a mixture of coliform organisms, Aerobacter, Paracolon, and also *Strep. viridans* and *Staph. aureus*, were cultured.

Sections from the mass showed a rapidly growing adenocarcinoma. This patient also had septicemia before death—culture from the heart muscle at post mortem yielded Paracolon and *E. coli*.

The gastric perforation was a terminal event.

Discussion

The five-year survival rate among patients with carcinoma of the stomach seen between 1953 and 1957 in northern Alberta was 12.9%. None of these 14 patients who perforated lived for five years; indeed the longest survivor was three years and five months. Only four lived more than one year. Free perforation of a gastric malignancy is a very serious complication of a bad disease.

Donaldson[9] reached the same conclusion in reviewing patients with free perforation of carcinoma of the colon; only 14% of those who survived resection lived for five years.

Fourteen patients perforated out of 1079 who had malignancy of the stomach: a perforation rate of 1.3%. The true rate is probably higher because cases are missed, particularly terminal cases not coming to post mortem. However, perforation of a gastric malignancy is a rare event.

Diagnosis is almost never made preoperatively and often not at operation because of the rarity of perforation of gastric malignancy. Except for the four terminal patients in this series, all were thought to have perforated peptic ulcers.

Eleven of the 14 patients were males. Of the tumours, one was a lymphosarcoma, one a leiomyosarcoma, the rest were carcinomas of the stomach. With the exception of a young man with perforated lymphosarcoma of the stomach, the patients tended to be older than those with perforated peptic ulcer: the average age was 64 years. Nine patients were operated on as an emergency: in four, the perforation was closed with an omental patch; in five, immediate gastrectomy was performed. Three of those closed with a patch later had a gastrectomy. Of the five patients who had immediate gastrectomy, three had a Billroth II resection and two a Billroth I.

The correct treatment appears to be gastrectomy, although omental patches held without leaking in four patients. Sutures used for placing the patch tend to pull out in the indurated tissue. If a patch is used a *biopsy should be taken from the edge of the ulcer*, and, if a pathologist is available, a frozen section done. Surprisingly, the early course was smooth in all nine patients treated surgically whether by patch or by gastrectomy. Three patients developed wound infections that were not serious. Thus, a gastrectomy can be done in the presence of gross peritoneal soiling with relative safety.

From a consideration of our patients all of whom showed rapid extension of disease, it is doubtful if a "second look" is justified. If a patch is used primarily, a gastrectomy should be done as soon as the patient's condition stabilizes and the peritonitis has settled down. In practical terms this would mean a second operation four to five weeks after the first one. This sequence was carried out in Case 7 (O.L.), and he lived for 41 months, the longest survivor. It was also carried out in Case 11 (F.A.) and he is still alive and well two years after operation.

Generalized peritonitis may occur in large carcinomas of the stomach without perforation. Perry and Shaw[10] describe 17 such patients coming to post mortem in whom no actual perforation could be found. In 12 of the 17 patients, the onset of peritonitis had been preceded by paracentesis or laparotomy but in five patients no obvious route for transmission of the infection was found. The following is the short history of such a patient.

S.M., a 67-year-old man, was admitted to hospital on October 28, 1961, with generalized abdominal pain of two weeks' duration and abdominal distension for three days. He had lost 40 lb. in the past three months. His condition was very poor and no surgery was done. Death occurred on November 10, 1961.

Autopsy showed extensive generalized peritonitis with 3000 c.c. of turbid fluid from which *Staph. aureus* and non-hemolytic streptococci were grown. There was a large fungating tumour in the upper portion of the body of the stomach measuring 15 x 15 x 4.5 cm., but no actual perforation. Section showed a well-differentiated adenocarcinoma.

Undoubtedly necrotic carcinomas provide a good culture medium for pathogenic bacteria, which can invade the peritoneal cavity in the absence of a gross perforation. Even without peritonitis, wound infection following gastrectomy for carcinoma of the stomach is twice as common as wound infection following gastrectomy for peptic ulcer.

In five of the 14 patients, gastric ulcers were thought to be benign both clinically and radiologically and were treated medically. They were in reality carcinomas; all gastric ulcers should be looked upon with suspicion.

Summary

A brief historical review of perforated gastric malignancy and our own experience with 14 such patients over a 13-year period are given. Because the condition is rare, the misdiagnosis of perforated peptic ulcer is routinely made. Treatment should be immediate gastrectomy: even in the presence of generalized peritonitis, this can be carried out with relative safety. The prognosis is poor—there were no five-year survivors, and only four patients lived more than one year.

I wish to thank the Record Departments of the Edmonton Branch of the Alberta Cancer Diagnostic Clinic, the University of Alberta Hospital, the Royal Alexandra Hospital, the Misericordia Hospital and the Edmonton General Hospital. Since most doctors have only seen a case or two in their surgical lifetime, I also thank the following doctors for making their cases available: W. S. Anderson, H. Hyde, G. L. Willox, H. T. G. Williams, R. J. Johnston, S. Kling, C. W. Weinlos, J. G. Kato —all of Edmonton, and R. M. Parsons of Red Deer and F. M. Smith of Camrose, Alta.

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Résumé

La perforation spontanée d'un néoplasme gastrique est une complication sérieuse d'une maladie grave. Chez 14 patients, aucun n'a survécu cinq années; 10 étaient morts dans les cours de la première année. Le patient qui a survécu le plus longtemps a vécu trois ans et cinq mois. Deux étaient encore vivants et apparemment bien à un an et deux ans.

La perforation est heureusement rare. Sur 1079 cas de cancer de l'estomac vus entre 1953 et 1965, il n'y eut que 14 cas de perforation, soit une proportion de 1.3%.

En raison de sa rareté, la perforation du cancer gastrique n'est quasi jamais diagnostiquée avant l'intervention. Sauf durant les phases terminales, ces perforations se présentent comme des ulcères gastro-duodénaux perforés.

Aucun des neuf patients opérés immédiatement n'est mort à l'hôpital. Au début tous les patients allaient bien. L'épiplooplastie a tenu chez quatre patients; mais le meilleur traitement opératoire est la gastrectomie immédiate, même en présence d'un péritoine fortement souillé. Cette opération peut être faite avec une sécurité relative.

RECESSION AND RELOCATION OF THE ENLARGED CLITORIS IN CONGENITAL ADRENOGENITAL SYNDROME*

R. J. SEYMOUR, M.D.† and R. A. H. KINCH, M.D., F.R.C.S.[C],‡ *London, Ont.*

CONGENITAL adrenal hyperplasia is a disorder of biosynthesis of the adrenal corticosteroids that results in the excessive production of androgens from the fetal adrenal cortex. It is due to a block in normal biosynthesis, especially of hydrocortisone.[1] The excessive androgen from the disordered fetal adrenal cortex induces masculinization of structures derived from the urogenital sinus, even in the presence of the Mullerian structures and the hormones from the fetal ovary. The newborn female infant presents with external genitalia and a urogenital sinus resembling the male to a varying degree. The appearance of the genitalia and the degree of virilization depends not on the time at which the fetal adrenals begin to secrete androgens abnormally, but on the quantity of androgen produced.[2] Therefore, the severity of this adrenal disorder can be roughly gauged by the extent of masculinization of the female external genitalia. Conversely, Childs, Grumbach and Van Wyk[3] believe that the degree of urogenital sinus deformity is influenced by the time of onset of excessive androgen production *in utero*.

The advent of sex chromatin testing has allowed a diagnosis to be made within a few hours of birth and should completely eliminate the tragic consequences of bringing up the genetic female as a "boy". We have noted in our series at Victoria Hospital, London, Ont. that the diagnosis is now made within weeks, whereas five to 10 years ago it was not made until the child was two to three years old.

INCIDENCE

It has long been known that adrenal virilism tends to recur within families and it is thought to be a genetic problem, the inheritance being by an autosomal recessive gene. In their classic study in 1956, Childs, Grumbach and Van Wyk[3] estimated that the incidence of congenital adrenal hyperplasia in the State of Maryland was 1:67,000. Prader[4] gives an incidence of 1:5041 in a closely observed population in the canton of Zurich. Between the years 1954 through 1964, 19 infants with congenital adrenal hyperplasia were admitted to the pediatric wards of the War Memorial Children's Hospital in London, Ont. Five of these were males and 14 were females. There were three sibling pairs in our series.

It might be thought that with the possibility of early diagnosis, i.e. within the first week by sex chromatin studies, and immediate treatment with cortisone, the clitoris would not become so greatly hypertrophied as to need subsequent plastic operation. We have not found this to be so; in fact, in our last three cases, all diagnosed within six weeks of birth, it has been obvious that surgical correction of the enlarged clitoris was essential. In this series of 14 infants with congenital adrenal hyperplasia, six are being maintained satisfactorily on cortisone with or without the addition of α-fluorone for the salt-losing syndrome. Of these six, two will probably need plastic repair. Five children have been treated by amputation of the clitoris and three by the operation of recession and relocation of the clitoris. This latter operation, originally described by Lattimer,[5] involves the removal of skin over the shaft of the enlarged clitoris, recession of the organ, partial amputation of the glans and relocation of the glans in a more appropriate position. It gives a more natural-looking perineum and also preserves the sensitive erectile tissue of the glans clitoris. We have now performed this operation on three children. The final result cannot be assessed because no patient has yet been followed into adult life.

PREOPERATIVE ASSESSMENT AND PREPARATION

The preoperative assessment of each of

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†Present address: Department of Obstetrics and Gynecology, University of Texas, Medical Branch, Galveston, Texas.

‡Department of Obstetrics and Gynecology, University of Western Ontario, Victoria Hospital and War Memorial Children's Hospital, London, Ont.

these patients must include a complete assessment of adrenal function. A pan-endoscopic examination of the vagina should be carried out to make sure that the cervix is present. The principal problem in these patients is to identify the external orifice of the urethra and to make sure that the posterior wall of this structure is not incised in the mistaken assumption that the incision is separating the labial fusion. For this reason a preoperative urogenital sinogram must be carried out because most of these girls have labial fusion, which obscures both urethral and vaginal orifices. The three girls in whom we carried out the Lattimer procedure were operated upon at 6½, 3¾ and 2½ years of age. Each child was well controlled with 25 to 37.5 mg. of cortisone or its equivalent daily. Two of the patients were also receiving 0.1 mg. of α-fluorone daily.

Preoperative preparation requires an increase in the dose of cortisone. The usual routine consisted of 100 mg. of hydrocortisone (Solu-Cortef) intramuscularly two to four hours preoperatively, 100 mg. intravenously during surgery and then 100 mg. every six to eight hours over the next 24 hr. Following this, the original dosage of cortisone was gradually resumed. This routine supported these patients excellently during the period of operation and the immediate postoperative period. Five hundred cubic centimetres of blood should be available before operation because the clitoris is a very vascular area and bleeding is sometimes excessive. The first girl needed 400 c.c. of blood and the second needed 300 c.c. The most recent case did not need blood replacement during the operation.

Technique

Under general anesthesia the child is placed in the lithotomy position and the urethral orifice is identified by a small catheter, size 6 to 8 French. The clitoris is then grasped and placed under tension so that the intended lines of incision can be outlined with a marking pen. An incision is made through the skin just behind the coronal sulcus of the glans clitoris, around its entire circumference. Another circumferential incision is made around the base of the clitoris; this incision is directed towards the

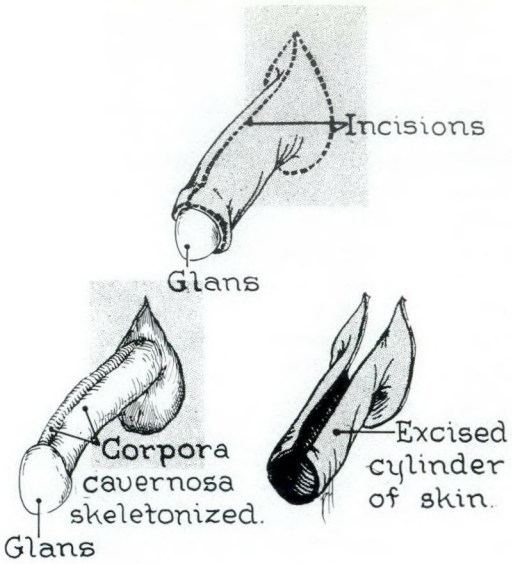

Fig. 1.—Outline of incisions.

mons pubis in the shape of an inverted V. (Fig. 1). These two circumferential incisions are then joined, removing the entire cylinder of skin covering the shaft of the clitoris and leaving the two corpora cavernosa "skeletonized" (Fig. 2). The suspen-

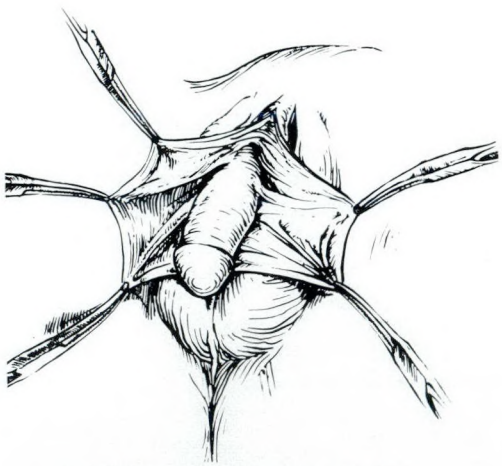

Fig. 2.—Denuding the clitoris.

sory ligament of the clitoris and other fibres that fix the base of the shaft to the pubis are divided so that the clitoris falls down towards the vestibule. The glans is then trimmed with sharp fine scissors or knife to an appropriate size (Fig. 3). It is at this stage that most operators seem to err on the conservative side. It is found that at least

one-third of the glans must be trimmed from each side of the glans. Strange as it may seem, there is very little bleeding during this part of the procedure.

A curved Kelly clamp is used to create a tunnel from the base of the shaft of the clitoris to a point just above the external urethral orifice where the glans of the clitoris normally lies. The Kelly clamp is then passed through this tunnel from below to grasp the glans and draw it through the tunnel to its new position just above the external urethral orifice (Fig. 4). The glans is sutured in its new position by four sutures of 4-0 plain catgut on a small, curved cutting needle. Hemostasis is achieved by ligating individual bleeding vessels and then placing transverse stitches of 4-0 plain catgut in the subcutaneous tissue above the base of the clitoris. This also draws the upper ends of the labia majora together. The skin is then closed with transverse stitches of 4-0 catgut or nylon on cutting needles. Finally, and with a catheter in the bladder to identify the urethra, a perineotomy is done as described by Howard Jones[6] (Fig. 5). The skin of the perineum is split in the midline and the vaginal mucosa split laterally. The posterior vaginal mucosa is drawn down and sutured to the skin edge of the new posterior forchette, thereby

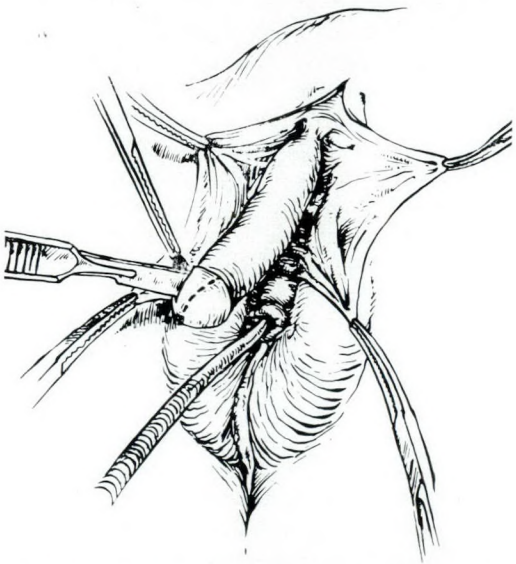

Fig. 3.—Freeing the clitoris and shaping the glans.

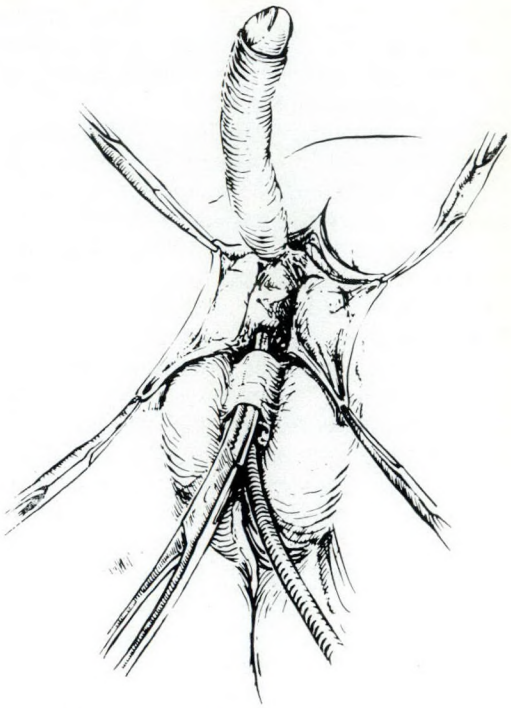

Fig. 4.—Creation of the skin tunnel.

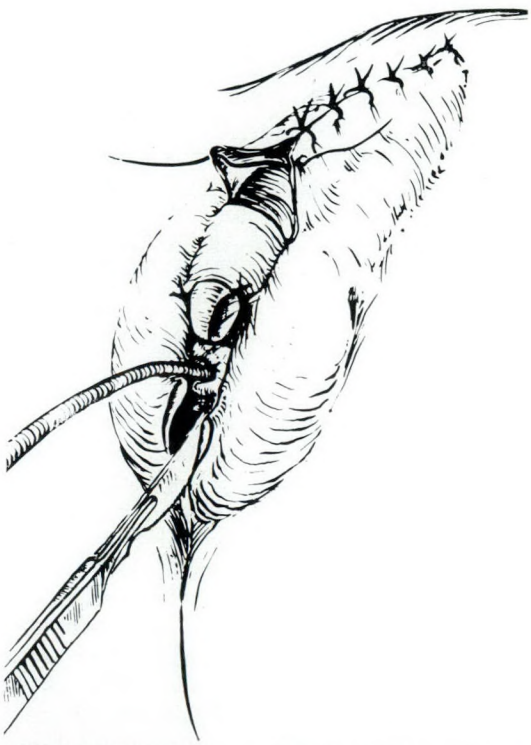

Fig. 5.—Clitoris drawn through skin tunnel; enlarging vagina.

opening up the vestibule. We are certain that in our cases a further operation will be necessary to enlarge the vaginal orifice at the appropriate time.

The patients have withstood this procedure quite well but, as has been indicated, in two cases blood replacement was necessary because of the vascularity of the area. Postoperative care is routine and the wounds heal very satisfactorily, the patients being discharged on about the tenth day. The main problem is urinary retention. One of the advantages of operating on these patients before they have achieved full toilet training is that bladder evacuation is quite involuntary and highly satisfactory. If the child is toilet trained she may need to be catheterized. This should be avoided as far as possible, partly because of the pain, but more important because of the possibility of cystitis and urinary-tract infection. The child is allowed up as soon as she understands that should not handle the operative area. Until then the arms should be restrained so that the child cannot damage the operative repair.

Postoperative Follow-up

Two of these children have been examined recently. The first patient was seen on November 28, 1965, 18 months after operation. Superficially, the external genitalia look quite normal. The skin of the labia and the transplanted mons looks different from the rest of the abdominal skin in this area. It is extremely loose and looks almost like scrotal skin. The obvious difference in texture will be obscured by the growth of pubic hair. The glans clitoris comes out at exactly the right place and is about ½ cm. wide. There is no obvious foreskin. The shaft of the clitoris can be felt quite easily and is not diminished in size. It is slightly sensitive. However, the skin over this is extremely loose and it does not appear that this little girl will have much discomfort should she sustain an erection of the clitoris. The vaginal orifice is patent and it will not need to be enlarged until she wants to get married. The other child was seen on December 20, 1965, three months after operation. Again the external genitalia are superficially quite normal and the same loose skin is seen over the clitoris. The urethra is seen as a separate opening and the vagina is the size of a lead pencil and probably will not need enlargement until marriage is contemplated.

Discussion

Since steroids with androgenic properties are now being carefully avoided in pregnant patients, fewer cases of iatrogenic clitoral hypertrophy are being encountered. It might be thought that clitoral hypertrophy secondary to congenital adrenogenital syndrome could be arrested if treatment with cortisone is started at birth and well maintained during infancy and childhood. In our experience this has not been the case. In the last three cases, the correct diagnosis was made within six weeks of birth and treatment with cortisone started, in one infant within three weeks of birth. However, in each of these children it was considered essential that the clitoris be operated upon to prevent embarrassment to the child. Ideally this operation should be done between the ages of two and four years, before the child takes an active interest in her genitalia and, perhaps more important, before she is exposed to other children at school. The decision as to whether this operation is needed should be made jointly by the pediatrician and the gynecologist. If there is any possibility that the child will "grow into the clitoris", the situation should be carefully watched and the operation avoided if possible. Lattimer[5] reported very good results in a series of 11 patients, but there was no long-term follow-up. So far we have been satisfied with the anatomical result of the operation and believe that the preservation of the erotically sensitive clitoris constitutes a much more physiological approach than amputation.

Summary

Three infants in whom the Lattimer operation—relocation and recession of the enlarged clitoris in congenital adrenal hyperplasia—was done are reported.

The operative technique is described in detail.

We believe that this operation is best carried out at about 2½ years of age when the clitoral hypertrophy is under good con-

trol with corticosteroids and before the child has evinced great interest in her own genitalia and particularly before she is the object of astonishment in the eyes of her peers.

The anatomical result of this operation has been extremely satisfactory.

The relocation of the clitoris with preservation of the erotically sensitive glans seems more physiological than amputation.

The authors wish to thank Professor J. C. Rathbun and Dr. Howard Valentine for the pediatric care of these cases and for permission to publish, and Mrs. Margaret Hutchinson, medical artist to Victoria Hospital, London, Ont., for the very clear illustrations.

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Résumé

Il est possible d'enrayer l'hypertrophie du clitoris qui est secondaire à l'hyperplasie surrénalienne d'origine congénitale, à condition que le traitement aux hormones surrénales soit appliqué dès la naissance. Il arrive cependant que le clitoris demeure assez grand pour représenter une gêne pour l'enfant. En dehors de l'amputation, un autre traitement consiste à reculer et à resituer le clitoris hypertrophié à un endroit plus approprié. Ceci permet d'obtenir une vulve d'apparance plus naturelle et de conserver sa sensibilité au tissus érectile de la portion distale du clitoris. Le résultat est satisfaisant du point de vue anatomique et cette opération est plus physiologique que l'amputation radicale.

Le moment idéal pour procéder à cette intervention se situe entre trois et quatre ans d'âge, soit avant que la curiosité de l'enfant pour ses organes génitaux externes ne soit éveillée et surtout avant que l'enfant ne risque d'être exposée aux regards de ses petites condisciples.

ERUCTATION AND EPIGASTRIC FULLNESS

Eructation has not received much attention in the literature. It is usually an involuntary act which may also be a conscious response to an ill-defined feeling of discomfort in the epigastrium. Intake of air depresses the diaphragm and the fundus of the stomach, at the same time widening the entry angle of the esophagus into the stomach. Abdominal muscles are contracted to increase intra-abdominal pressure and the spine is extended to elongate the esophagus. The cardia becomes funnel-like from traction and the intraesophageal pressure decreases. If there is sufficient stomach gas and a pressure gradient exists, the cardia should open and eructation occur. Effervescent drinks assist by increasing the volume of fundal gas and even a small volume of swallowed air deliberately taken into the stomach may be sufficient to make an eructation possible.

It is unusual to see significant collections of stomach gas in patients complaining of epigastric fullness. Gas appears to accumulate only in the fundus above the level of the cardia as it does in others. What then produces this symptom? Elevation of the diaphragm from any cause, including a gas-filled fundus, can produce discomfort in the epigastrium or left hypogastrium; symptoms occur in pregnancy, ascites, gaseous distension of the splenic flexure (splenic-flexure syndrome), gaseous distension of other parts of the bowel and after therapeutic pneumoperitoneum. Discomfort may occur after a heavy meal followed by sitting in a flexed position, when abdominal contents are compressed against the diaphragm. Relief is obtained by standing or extending the spine.

In obesity the diaphragm is relatively high due to accumulation of omental fat and when weight is lost the diaphragm assumes a lower level. Symptoms of epigastric fullness are common in such persons. Corsets always accentuate the discomfort.

Elevation of the left cupola of the diaphragm raises the fundus relative to the cardia and increases the volume of accumulated gas, although the additional amount probably is not significant. However, it also closes the cardia more tightly making eructation more difficult.—Hood, J. H.: Clinical considerations of intestinal gas, *Ann. Surg.*, **163:** 364, 1966.

LES TROUBLES DU METABOLISME ACIDO-BASIQUE EN CHIRURGIE GENERALE*

J. N. NORMAN, M.D., Ph.D. et D. T. CARIDIS, M.D., *Aberdeen, Ecosse.*

Il n'est que depuis quelque temps, que certains auteurs[1-3] ont reconnu l'importance de diagnostiquer rapidement et de corriger précisément les troubles du métabolisme acido-basique survenant pendant et après la chirurgie à cœur ouvert. Il a de plus été démontré récemment que des troubles graves de l'équilibre acido-basique se produisent souvent en chirurgie générale et que le traitement adéquat de ces modifications peut sauver la vie de beaucoup de malades.[4-6] Le développement de méthodes susceptibles de déceler et de traiter rapidement et précisément les troubles acido-basiques devrait conduire à faire un plus grand usage de cette donnée dans la pratique clinique courante.[7, 8]

Acidose Métabolique

En chirurgie générale, on rencontre habituellement une acidose métabolique dans deux circonstances: (1) L'acidose à début aigu; celle-ci est associée à des phénomènes dramatiques tels que l'arrêt cardiaque, le choc traumatique ou une grave hémorragie; une fois la cause primaire traitée, et pourvu qu'aucune lésion cérébrale irréversible ne se produise, la correction de l'acidose à ce stade permettra une guérison complète. (2) L'acidose progressive; celle-ci est généralement associée à une perte continue de bases, comme cela peut se produire dans une fistule pancréatique; c'est ordinairement un problème métabolique compliqué, et souvent la seule manifestation de l'acidose croissante est la détérioration progressive de l'état général non expliquée par les examens courants. Aussi la correction de l'acidose amène-t-elle une amélioration clinique notable qui peut se maintenir tandis que l'on traite la cause sous-jacente.

Observations

Cas 1.—Malade opéré pour une coarctation aortique. Au cours de l'intervention deux épisodes d'hypotension grave ont lieu; quatre heures après l'intervention, le malade n'a pas repris connaissance, les pupilles sont dilatées, il ne réagit pas à la douleur. La dépression respiratoire est telle qu'il faut pratiquer la respiration artificielle. On pense alors que cet état est dû à une anoxie cérébrale qui s'est formée pendant les épisodes d'hypotension, mais l'étude de l'équilibre acido-basique montre l'existence d'acidose métabolique modérée (Tableau I). L'acidose est alors corrigé par l'administration de 152 mEq./l. de bicarbonate de soude; une respiration spontanée suffisante et une réponse à la douleur réapparaissent immédiatement. La conscience ne revient pas cependant complètement, et le malade reçoit de l'oxygène sous deux atmosphères pendant quatre heures. La guérison est complète; nous pensons que dans ce cas les troubles étaient dus à une acidose métabolique, conséquence de l'anoxie causée par l'hypotension.

Cas 2.—Vagotomie et pyloroplastie pour ulcère duodénal. Trois jours plus tard, le malade se plaint d'épigastralgies; le quatrième jour survient un collapsus après apparition d'une douleur abdominale généralisée. On découvre alors une grave acidose métabolique, et pour permettre une laparotomie immédiate, on corrige celle-là à l'aide de 300 mEq./l. de bicarbonate de soude (Tableau I). L'opération révèle l'existence d'une embolie de l'artère mésentérique supérieure associée à une modification d'un segment considérable d'intestin grêle. Tout était en voie de régression spontanée et nous supposons que pendant la période d'ischémie, l'intestin avait été la source de l'acidose sévère.

Cas 3.—Gastrectomie d'urgence pour melaena persistant. A l'intervention, il est difficile de mobiliser le duodénum en raison d'une réaction inflammatoire de voisinage. La fermeture duodénale adéquate est impossible et on laisse une sonde de Pezzer dans le duodénum. Le 18ème jour on enlève le drain et la fistule persiste pendant deux semaines. Le 19ème jour, le malade devient asthénique et dyspnéique et on découvre une acidose métabolique. On lui donne alors 200 mEq./l. de bicarbonate de soude: son asthénie disparaît et son rythme respiratoire redevient normal. La déperdition des sucs duodénaux alcalins, dont la teneur en bicarbonate de soude est de 13

*Département de chirurgie de l'Université d'Aberdeen, service du Professeur George Smith, Aberdeen, Ecosse.

TABLEAU I.—CONDITIONS DANS LESQUELLES ON A DÉCOUVERT UNE ACIDOSE MÉTABOLIQUE

No. de cas	*Age (ans)*	*Temps (heures)*	*pH*	*pCO_2 (mm. Hg)*	*Standard bicarbonate (mEq./l.)*	*Réserve alcaline (mEq./l.)*	*Bicarbonate de soude remplacer (mEq./l.)*
1	19	0	7.28	38	17.6	− 7.8	152
		12	7.39	43	24.5	+ 1.0	
2	55	0	7.24	25	13.2	−15.0	300
		4	7.48	34	25.5	+ 3.5	
3	55	0	7.27	30	15.6	−11.3	200
		8	7.37	38	21.0	− 1.5	

mEq./l., avait entraîné l'acidose métabolique (Tableau I) responsable de la détérioration progressive de l'état du malade. La correction immédiate de l'acidose amène une rapide amélioration clinique.

ALCALOSE MÉTABOLIQUE

L'alcalose métabolique est fréquente en chirurgie générale à la suite d'une déperdition du contenu gastrique. On la voit donc le plus ordinairement dans la sténose du pylore;[3] dans cette affection on a montré que le niveau de l'alcalose était en rapport plus étroit avec l'état général du malade qu'avec le chiffre des électrolytes du sérum. Dans ces cas, pourtant, l'alcalose, n'est qu'une manifestation d'un trouble métabolique complexe comprenant la déshydration, l'hypokaliémie, et l'hypochlorémie. Dans la majorité des cas il y a peu d'avantages à traiter l'alcalose *in se*, mais l'étude de l'équilibre acido-basique fournit un indice sûr de l'efficacité de la réhydration. Dans beaucoup de cas, l'alcalose ne peut pas être corrigée par le remplacement des liquides, mais elle l'est rapidement par la suppression chirurgicale de l'obstruction: dans la Fig. 1, on voit les résultats biochimiques obtenus chez un malade de ce type. Cependant, à moins d'entreprendre la réhydratation avant l'opération pour sténose serrée du pylore, il y a un danger d'accentuer l'alcalose à la phase post-opératoire immédiate par la réponse métabolique au trauma.[9] Dans notre série, les malades qui se présentaient avec un taux standard de bicarbonate de soude à plus de 33 mEq./l. et une augmentation de la réserve alcaline à plus de 10 mEq./l. avaient besoin de recevoir des liquides et des électrolytes, en particulier du potassium, pour que l'opération puisse être effectuée en toute sécurité. Cela a été fait la nuit précédant l'opération. Chez ceux dont le taux standard des alcalins était plus grand que 20 mEq./l., l'état général était si grave qu'il a fallu des soins intenses pendant 48 heures ou davantage avant qu'ils ne soient aptes à être opérés. Le dosage du pH sanguin dans ces cas n'indique pas fidèlement la gravité des troubles, à cause du phénomène de compensation respiratoire: chez tous nos malades le pCO_2 était tel que le pH ne s'élevait pas au-dessus de 7.59 même dans les alcaloses métaboliques les plus graves.

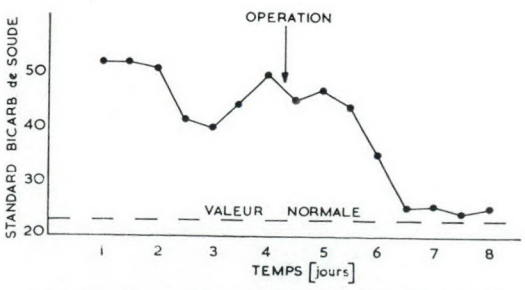

Fig. 1.—Correction rapide de l'acalose après intervention chirurgicale.

Grâce à cet examen chez nos malades chirurgicaux où nous soupçonnions une perturbation métabolique quelconque, il nous a été possible de montrer qu'un certain nombre d'entre eux presentaient une alcalose métabolique qui était associée la plupart du temps à une déperdition des liquides gastro-intestinaux (dus à des fistules qui siégaient à divers endroits du tube digestif) ou à une hypokalémie. Le Tableau II montre des exemples de ce type de malades ainsi que le degré de leur alkalose. Cette alkalose était elle-même considérée

TABLEAU II.—Conditions dans lesquelles on a découvert une alcalose métabolique

No. de cas	*Diagnostic*	*K^+ du sérum (mEq./l.)*	*pH*	*pCO_2 (mm. Hg)*	*Standard bicarbonate de soude (mEq./l.)*	*Réserve alcaline (mEq./l.)*
1	Tumeur du Conn	2.2	7.60	70	44	+17.4
2	Thrombose de la veine portale	4.0	7.50	37	29	+ 8.5
3	Ulcères multiples du gros intestin	3.5	7.47	57	32	+ 9.4
4	Tumeurs papillomateuses de Villi du gros intestin	2.4	7.50	37	28	+ 6.0
5	Défaut du septum atrial	3.8	7.36	51	27	+ 5.0
6	Syndrome de loope afférente	3.0	7.40	61	31	+11.0

comme la manifestation isolée d'un trouble métabolique complexe et elle n'a pas été traitée d'un façon spécifique bien que le contrôle acido-basique ait fourni un indice utile de l'efficacité du traitement par remplacement des liquides et des électrolytes. Par opposition le traitement de l'acidose métabolique exige promptitude et énergie si l'on veut éviter de dangereuses conséquences au niveau du myocarde.[10]

Une autre origine de l'alcalose métabolique qui n'a pas en général été notée autrefois, est la transfusion massive de sang ACD: à la suite de cette thérapeutique on peut enregistrer une augmentation des bases jusqu'à +9, même deux semaines après la transfusion. Il n'y a pas alors de troubles métaboliques associés et l'alcalose est due au citrate.

Conclusion

Pendant une période de 18 mois, les auteurs ont découvert l'existence d'une acidose métabolique chez 60 malades: chez 40 d'entre eux elle fut corrigée avec du bicarbonate de soude. Dans beaucoup d'autres cas, une alcalose métabolique a constitué une découverte fortuite: l'évolution de cette perturbation a donné une bonne impression de l'efficacité du traitement par les liquides et les électrolytes. Nous pensons donc que dans les cas où une grave atteinte de l'état général ne peut être expliquée par les examens courants, il est formellement indiqué d'apprécier l'équilibre acido-basique.

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Summary

During a period of 18 months, 60 patients with metabolic acidosis have been studied. In 40 of them the metabolic acidosis had to be corrected with intravenous administration of sodium bicarbonate. This was followed by rapid amelioration of the clinical state of the patients. In a number of cases the metabolic alkalosis had to be corrected with electrolytes and fluid replacement. We conclude that in cases of sudden grave clinical deterioration, where an explanation cannot be obtained by the routine examinations, one should appreciate the value of the assessment of the acid-base equilibrium.

RECURRENCE AT THE OPERATIVE SITE AND ITS RELATION TO CANCER GROWTH IN MICE*

JOHN A. McCREDIE, M.B., M.Ch., F.R.C.S.[Eng., Edin. & C] and
W. RODGER INCH, B.Sc., Ph.D., A. Inst. P., *London, Ont.*

TUMOURS still recur frequently at operative sites even though criteria for selecting patients to be treated by excision have been better defined in recent years and surgical techniques have been improved.[1] Results obtained by the adjunctive use of chemotherapy or radiotherapy have generally been disappointing. However, it is possible that maximal benefit has not been obtained because the mechanism by which recurrences develop is poorly understood.

Shimkin *et al.*[2] determined the rate of growth of cancer of the human breast from the interval between the onset of symptoms or the recognition of a mass, and operation; they found that local recurrence was earlier and more frequent in those with rapidly growing tumours. It has also been shown that the survival rate was lower among patients treated by radical mastectomy when the tumours had stellate or serrated, rather than smooth or nodular contours; the relation of these gross characteristics to local recurrence was not determined.[3] Cancer can be graded microscopically according to the features of the malignant cell, its relation to normal tissues and the type of cellular reaction it induces; however, such grading is often difficult because the appearance may vary in different regions of the tumour. Pawlias, Dockerty and Ellis[4] found that local recurrence after radical mastectomy was not related to the grade of malignancy. The extent of local spread beyond the tissue of origin and to neighbouring lymph nodes are the most important factors.[5] Local recurrence is uncommon in patients with Stage A (Columbia Clinical Classification) cancer of the breast, occurs in about 10% with Stage B tumours and is so frequent in those with more advanced disease that excision is rarely indicated. Recurrence develops at the operative site from "free" cancer cells occurring singly or in clumps, or from residual processes of tumour. Superficial contamination may occur from divided blood vessels or lymphatics containing tumour emboli after taking a biopsy or cutting into a tumour, or after opening a hollow organ containing the growth. Malignant tissue may be left at the operative site as a result of spread "in continuity" into the interstitial tissues, within normal cells, by permeation of lymphatics, blood vessels and ducts, or infiltration of serous cavities and lumen of viscera. Conflicting views are held on the importance of these mechanisms. If recurrence occurs from "free" cells, many should be found in wound washings, and the topical application of chemicals could reduce recurrence. If residual tumour is the more frequent cause of local recurrence, then few cells are likely to be found in the washings and the local use of chemicals, which do not penetrate the surface, will not reduce recurrence. The finding of single cancer cells in washings from patients was initially thought to be related to recurrence but this has not been confirmed[6] and irrigation of the operative site has not reduced recurrence. In the only investigation suggesting that irrigation was of value, Smith *et al.*[7] found that, in patients with cancer of the cervix, local recurrence was related to the finding of cells and the applicaton of proflavine was of value, but only in those with negative washings. The clinical use of wound irrigation has been stimulated by the apparent positive results in experiments in animals, which did not closely reproduce the conditions in patients. Only superficial contamination has been produced; an incision has been made into the subcutaneous tissues, a solution containing cancer cells placed in the wound and after one hour the area has been irrigated with a chemical.[8] In addition, an allogeneic tumour, which is more sensitive to chemicals, has generally been used.

*From the Departments of Surgery and Therapeutic Radiology, University of Western Ontario, and The Ontario Cancer Treatment and Research Foundation, London Clinic, London, Ont.

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We have demonstrated that irrigation of the site of excision of established mammary tumours in C3H mice with solutions containing mechlorethamine (nitrogen mustard, HN_2) or a brand of monoxychlorosene (Clorpactin XCB)[9] or a single dose of x-rays (2000 rad) to the operative site one day after excision[10] had no effect, but administration of the same dose of radiation to the tumour and its bed six days before operation decreased local recurrence.[11] The rate of cancer growth, the microscopic appearance of the tumour and its bed and the presence of cancer cells in washings from the operative site were studied in an attempt to explain these observations.

Method

Two spontaneous mammary carcinomas occurring in adult C3H/HeJ mice* were excised using an aseptic technique; small pieces (5 ± 2 mg.) containing 3×10^4 viable cancer cells were implanted by trochar into the dorsal subcutaneous tissues of isologous 10- to 12-week-old (20 g.) female offspring. Cells were considered viable if they did not stain with eosin. The time of appearance of the tumours was noted, the rate of growth was determined from measurements of two diameters using a vernier caliper and the degree of fixation to the deep fascia was assessed at the time of operation. The isoimplants were excised aseptically at a size (2.0 ± 0.4 cm.^{2}) that was followed by a high incidence of local recurrence; they were weighed wet and again after 72 hr. at 37°C. in a vacuum oven. The operative site was irrigated with 100 ml. of saline (0.9 g./100 ml.), the first 40 ml. collected and centrifuged, a constant volume of the sediment placed on a slide and fixed and stained using the Papanicolaou technique; the slides were then examined for the presence of cancer cells. The operative site was closed using a continuous silk suture and the animals observed for local recurrence during 100 days. Those animals in which recurrence developed, and survivors at 100 days were killed and examined for recurrence and distant metastases. A number of mice were killed, the tumour excised, the operative site washed with saline and histological sections of the implant and the site of excision were prepared.

Spontaneous mammary tumours in C3H mice were not used because they are often multiple, superficially placed, variable in position and seldom recur after excision. A large number of single, isologous implants was obtained for this study by growing the spontaneous tumour as described, and there was a high incidence of local recurrence after excision when the tumour was implanted deeply into the subcutaneous tissues. The blood supply[12] and method of spread is probably similar in spontaneous and transplanted C3H tumours.

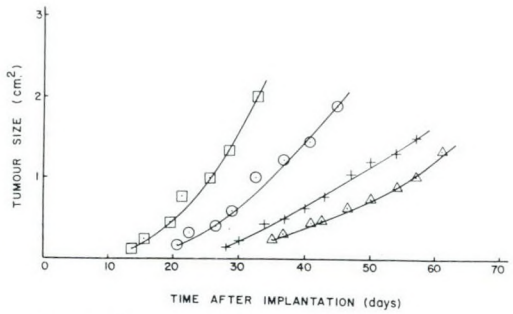

Fig. 1.—Growth curves of C3H isoimplants that appeared at 13 (□), 21 (○), 28 (+) or 36 (△) days after implantation. Points represent the mean values for five tumours.

Results

Tumours became palpable from 13 to 36 days after implantation and those that

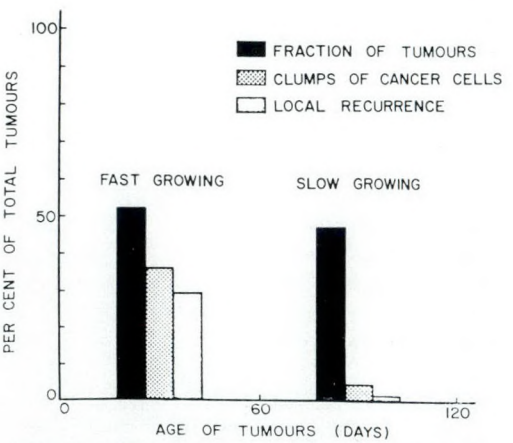

Fig. 2.—"Fast-growing" C3H isoimplants had a higher incidence of clumps of cancer cells in wound washings and of local recurrence than those that grew slowly.

*Obtained from the Jackson Laboratory, Bar Harbor, Maine.

TABLE I.—Local Recurrence after Excision of the C3H Isoimplant in the Mouse

Age of tumour at excision	No. of animals	Body weight at excision (g.)	Tumour at excision: Fixation to deep fascia (%)	Tumour at excision: Size (cm.²)	Tumour at excision: Wet weight (g.)	Tumour at excision: Dry/wet weight (%)	Clumps of cancer cells in washings (%)	Local recurrence within 100 days (%)
Less than 60 days ("Fast growing")	34	23 ± 2.6	23	2.0 ± 0.47	1.2 ± 0.43	21 ± 3.7	55	56
More than 60 days ("Slow growing")	31	25 ± 2.5	0	2.1 ± 0.30	1.1 ± 0.30	20 ± 2.1	13	3
Total	65	24 ± 2.3	12	2.0 ± 0.39	1.2 ± 0.10	20 ± 3.4	40	31

appeared first grew most rapidly (Fig. 1). They reached a size of 2.0 ± 0.4 cm.² within 18 to 120 days and were described as "fast growing" when the time from implantation was 60 days, and "slow growing" when the interval was longer (Table I and Fig. 2). Fixation to the deep fascia occurred in 23% of the animals with "fast growing" but in none with "slow growing" tumours. The mean wet tumour weight (1.2 ± 0.10 g.) and the ratio of dry-to-wet weight (20%) were the same for the two groups. Clumps of cancer cells were found in the wound washings in 55% of the animals with rapidly growing tumours but in only 13% of those that grew slowly ($P < .01$); they were present in 88% of those with tumours that were fixed. The incidence of local recurrence was related to the rate of tumour growth; it was 56% in animals with rapidly growing and 3% in those with slowly growing tumours ($P < .01$).

We have observed that untreated C3H isoimplants do not regress after reaching a diameter of 0.5 cm.; they eventually ulcerate, the host loses weight, develops anemia and dies within several days. When the primary tumour is not treated, metastases are found at autopsy in the lungs of about one-third of animals but rarely cause death; in the present study, only 12% of animals that developed local recurrence had metastases.

It was not possible to grade the tumours accurately because the histologic appearance varied in different regions of the same tumour. Implants that grew slowly had smooth surfaces (Fig. 3) while the deep surface of those that grew rapidly was often irregular (Fig. 4). Processes were

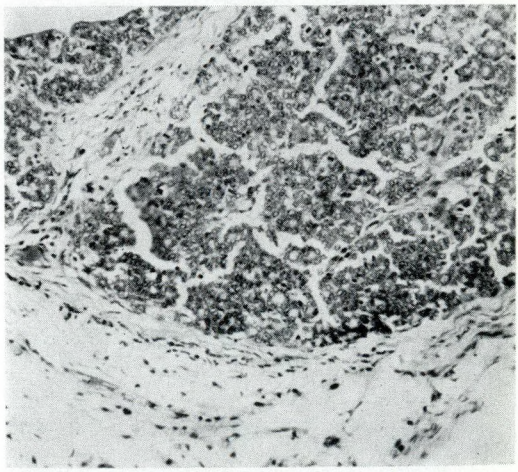

Fig. 3.—"Slow-growing" C3H isoimplant 70 days after implantation, showing smooth, deep surface of the tumour and no processes infiltrating the normal tissues. (H & E)

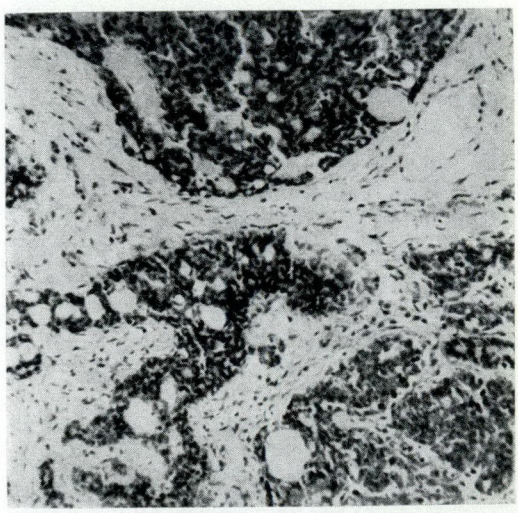

Fig. 4.—"Fast-growing" C3H isoimplant 30 days after implantation, showing processes of tumour infiltrating the normal tissues. (H & E)

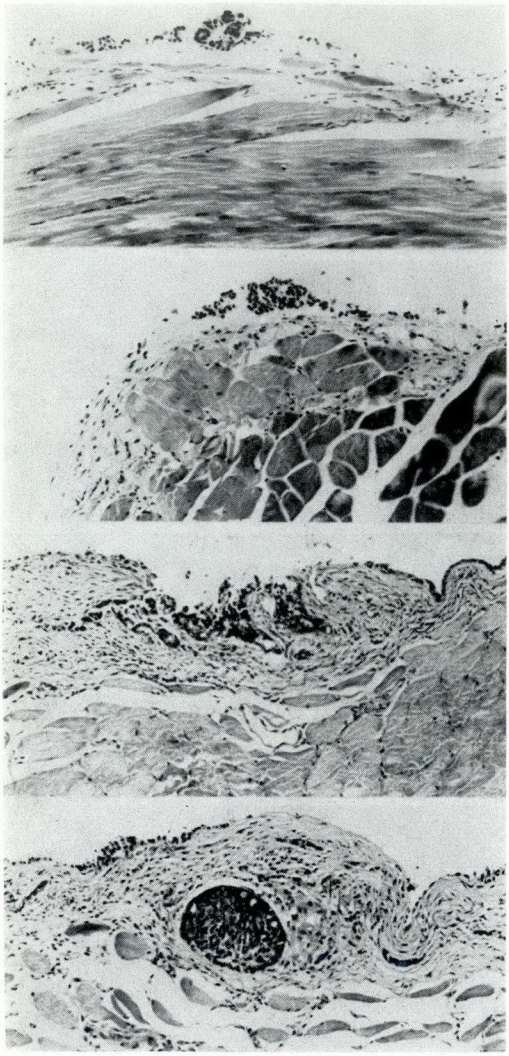

Fig. 5.—Residual tumour found at the site of excision of "fast-growing" C3H isoimplants. (H & E)

seen at the operative site in the interstitial tissues and permeating capillaries, only in animals with rapidly growing tumours (Fig. 5); in animals with fixed tumours, cancer cells were sometimes observed in the underlying muscle. Lymphocytes and plasma cells were occasionally seen in the tumour and its bed. It was difficult to state with certainty whether isolated cells found in wound washings were benign or malignant (Fig. 6A). Groups of cells, however, could be recognized with greater accuracy (Fig. 6B) and they resembled clumps found in smears taken directly from the cut surface of the tumour (Fig. 6C).

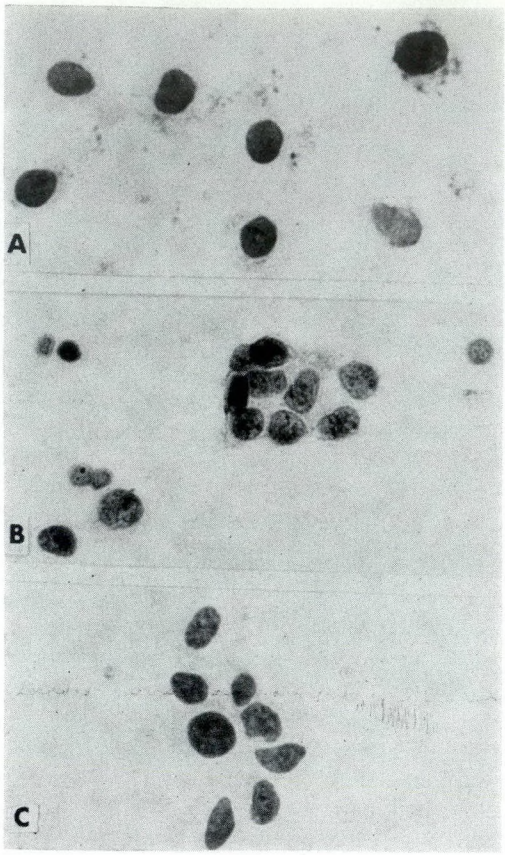

Fig. 6—(A) Single cells found in wound washing. Identification as benign or malignant is difficult (Papanicolaou). (B) Clump of cancer cells found in wound washing (Papanicolaou). (C) Clump of cancer cells in smear taken from the cut surface of the tumour (Papanicolaou).

Discussion

The results of this study showed that about one-half of the mice with rapidly growing isoimplants had clumps of cancer cells in wound washings and developed local recurrence after excision, while animals with slowly growing tumours seldom had clumps of cells or recurrence. Processes of tumour were seen in continuity with the deep surface of the implant and at the operative site in those with rapidly growing tumours; their division could readily have given rise to the clumps of cells seen in the washings and persistence of tumour at the site of excision to local recurrence. This would explain the failure of topically applied chemicals, which act only on surface cells, to reduce recurrence and the effectiveness of concentrated preoperative radia-

tion, which penetrates the tissues and damages the cancer cells directly, and through its effect on the tumour bed. The inability to identify with accuracy single cancer cells in the wound washings was in agreement with the experience of Weinlos, Macdonald and Taylor[13] who found that 13% of patients found to have positive washings did not have cancer.

The results are consistent with the clinical observations that prognosis is worse in patients with tumours that are growing rapidly and have irregular surfaces. They also support our findings in a previous study[9] that irrigation of the site of excision of C3H isoimplants with solutions containing Clorpactin XCB or nitrogen mustard did not reduce recurrence and strongly suggest that recurrence develops from residual tumour and not from superficial contamination of the operative site with cancer cells. The topical application of chemicals is therefore not indicated but the adjunctive use of radiation before operation in patients who are likely to develop local recurrence is probably of value. Damage by ionizing radiation depends largely on the concentration of oxygen in the cell and therefore radiotherapy will be less effective after operation when peripheral processes primarily responsible for recurrence are anoxic due to an impaired blood supply. A conventional large dose of radiation is not necessary and the concurrent use of hyperbaric oxygen is unlikely to improve results further since an increase in the oxygen tension of well-oxygenated tissue produces little increase in radiosensitivity.[14]

It is not clear why the rate of growth of the tumours varied so much. The spontaneous C3H tumour is an adenocarcinoma that contains cells at various stages of differentiation, therefore implants taken from areas in the tumour containing a high proportion of anaplastic cells would be expected to grow more rapidly. The generation time of malignant cells depends on their proximity to blood vessels and therefore those originating from areas at a low oxygen tension would be expected to appear late. Their subsequent rate of growth, however, depends largely on the local blood supply. Similarly the number of cells implanted could affect the time of appearance of the tumours but not their subsequent rate of growth. The fact that two donor tumours were used was not important because each gave rise to the same proportion of rapidly and slowly growing implants. The inocula were sterile and wound infection did not occur. The offspring belonged to the same inbred strain, were the same sex and body weight and were kept under similar conditions. The tumour-host relation was isologous because all of the implants grew, regression did not occur, and few lymphocytes and plasma cells were seen in the tumour and its bed. A difference in host resistance and selective growth of rapidly or slowly growing clones of implanted cells, depending largely on the area of the tumour from which the implant was taken, were the most likely causes of the variation in the rate of tumour growth.

Summary

Cancer recurrence at the operative site was studied using established isoimplants of the spontaneous mammary carcinoma in C3H mice. Tumours appeared at different times after implantation into the subcutaneous tissues; those that appeared early grew rapidly and some became fixed to the deep fascia while those that developed late grew slowly and remained mobile. The implants were excised when they reached a size (2 cm.^{2}) that was followed by a high incidence of local recurrence. Animals with rapidly growing tumours more often had clumps of cancer cells in wound washings, a tumour surface that was irregular, residual processes of cells at the operative site, and a high incidence of local recurrence. Those with slowly growing implants did not have processes, seldom had clumps and rarely developed recurrence. Division of the processes gave rise to the clumps of cancer cells seen in the washings and the persistence of these processes in the tissues at the operative site was the cause of recurrence. The results support the clinical observation that rapidly growing tumours, particularly those with irregular surfaces, are more likely to recur after excision. They are in agreement with conclusions reached from previous work in this laboratory, which suggested that irrigation of the

operative site with chemicals is not indicated and that a single large dose of radiation given to the tumour and its bed six days before operation may reduce local cancer recurrence.

The authors are indebted to the late Dr. R. G. S. Malone, who interpreted the cytology and to Mistresses R. Stodolski, M. Alderson and Y. Fotheringham, who assisted with the experiments. Mr. D. Pulham prepared the photographs.

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Résumé

On a étudié les rechutes du cancer au siège opératoire; pour ce faire on a utilisé des isoimplants de cancer mammaire spontané chez des souris C3H. Les tumeurs sont apparues à différents moments après l'implantation dans le tissu souscutané; celles qui apparaissaient tôt eurent une croissance rapide et certaines se fixèrent au fascia profond, tandis que celles qui se développaient plus tard eurent une croissance lente et restèrent mobiles. Les implants furent excisés une fois qu'ils eurent atteints une dimension (2 cm.^{2}) telle qu'elle était suivie par une grande fréquence de rechute locale. Les animaux porteurs de tumeurs à croissance rapide présentaient plus souvent des amas de cellules cancéreuses dans les lavages de plaies, une surface tumorale irrégulière, des résidus cellulaires au siège opératoire et manifestaient une forte tendance aux rechutes. Les souris chez lesquelles les implants croissaient lentement, n'avaient pas de résidus cellulaires, rarement d'amas de cellules et présentaient peu de rechutes. La division des résidus cellulaires a donné naissance aux amas de cellules cancéreuses observées dans les lavages et la persistance de ces résidus dans les tissus au siège opératoire a été la cause des rechutes. Ces résultats confirment l'observation clinique que les tumeurs à croissance accélérée, particulièrement celles qui ont une surface irrégulière, ont une plus forte tendance à la récidive après excision. Ils sont d'accord avec les conclusions des travaux faits antérieurement dans ce laboratoire, conclusions tendant à démontrer qu'il n'est pas indiqué d'irréguer le siège opératoire avec des produits chimiques et qu'une seule forte dose de radiation appliquée à la tumeur et à son siège six jours avant l'opération permet de réduire la fréquence de la rechute locale.

NON-PENETRATING ABDOMINAL TRAUMA*

R. A. H. McLEOD, M.D. and D. R. BROWN, M.D., F.R.C.S.[C], *Ottawa, Ont.*

IN RECENT years several reports have described the problems encountered in the management of non-penetrating abdominal trauma. Since World War II, the mortality rate from penetrating wounds has shown a steady decline due, in most part, to improved methods of resuscitation and treatment.[1] These improvements, however, have failed to cause any marked drop in the mortality rate from blunt trauma to the abdomen. While the mortality rate from penetrating wounds is 7 to 10%, it is still 10 to 30% from blunt trauma.[2] The explanation of this discrepancy seems to lie in the ease with which the initial diagnosis is made in penetrating wounds. Unlike penetrating wounds, injuries caused by blunt trauma, even when severe, are frequently not obvious. Physical findings may be lacking, obvious injuries to other parts of the body may obscure the abdominal trauma and, if the associated injury requires an emergency operation under general anesthesia, the signs and symptoms of intra-abdominal injury may be further suppressed.[3, 4] Under these circumstances an early diagnosis may be virtually impossible. To quote one author,[5] "The surgeon must be guided more by general considerations of the clinical picture than by the presence or absence of any particular sign."

Until recently less than 0.1% of admissions to a general hospital were due to blunt abdominal trauma;[6] however, the automobile is pushing this figure upwards.

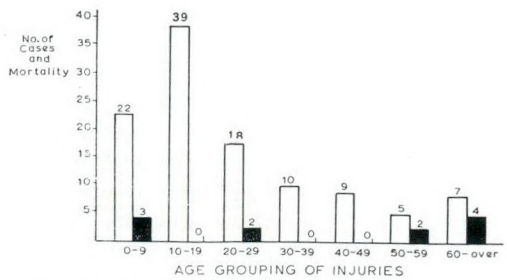

Fig. 1.—Graph illustrating age grouping and mortality rate.

*From the Department of Medical Education and Research, Ottawa Civic Hospital, Ottawa, Ont.

Automobile accidents account for at least 50% of these injuries[7] and with 12% increase in automobile accidents each year,[8] we can expect to see more and more patients with blunt abdominal trauma admitted to our hospitals. The object of the present communication is to point out some of the problems that can be encountered in this group of patients.

MATERIAL

This report is based on an analysis of 110 patients treated at the Ottawa Civic Hospital over the five-year period, January 1960 through December 1964. Only those patients with proved intra-abdominal injuries were included. There were 86 males and 24 females, a ratio of almost four to one. There were 11 deaths, a mortality rate of 10%. The highest incidence of injury occurred in the 10- to 30-year age group (Fig. 1); however, the mortality rate was significantly higher at the extremes of life (Table I). Vehicular accidents accounted for both the highest number of injuries and the greatest mortality (Table II).

TABLE I.—AGE GROUPING AND MORTALITY RATE

Age (yr.)	*Patients*	*Deaths*	*Mortality rate (%)*
Under 10	22	3	14
10 to 60	81	4	5
Over 60	7	4	57

TABLE II.—MODE OF INJURY

Injury	*Patients*	*Deaths*	*Mortality rate (%)*
Automobile accidents	64	9	14
Falls	11	1	9
Struck by an object	10	1	10
Blows	5	0	0
Athletics	20	0	0

A review of Table III demonstrates that trauma to other organ systems is frequently associated with intra-abdominal injury; 61 patients (55%) had associated injuries. Thoracic involvement was common (26 cases); significant head injuries occurred

TABLE III.—Associated Injuries

Injury	*Patients*	*Deaths*	*Mortality rate (%)*
Head	4	0	0
Head and thorax	7	4	57
Head and extremities	13	2	15
Thorax	11	1	9
Thorax and extremities	8	2	25
Extremities	18	2	11

in 24. The highest mortality rate occurred in those patients with the combination of thoracic, head and abdominal injuries. When all three areas were involved, the death rate was 57%. The overall mortality rate when extra-abdominal injuries were present was 16%. On the other hand, there were no deaths when only intra-abdominal organs were involved.

It is noted in Fig. 2 that solid viscera (92 cases) were damaged more commonly than hollow organs (33 cases). The kidney seemed to be most vulnerable but usually received only minor trauma, as indicated by the fact that only nine of 51 cases required surgery. In each instance the surgery was performed to control bleeding caused by disruption of the kidney parenchyma or the renal pedicle.

Twenty-two patients had rupture of the spleen. In five of them there was a delay in diagnosis of more than eight hours. All of this latter group had injuries to extra-abdominal organs, which had masked the abdominal injury. Delay in diagnosis was the major factor in the death of one patient.

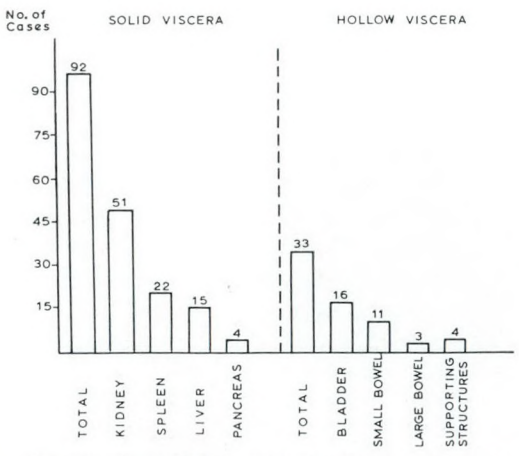

Fig. 2.—Graph illustrating distribution of injuries to abdominal viscera.

Delayed rupture of the spleen occurred in one patient. Except for a fractured rib, he had been asymptomatic at time of injury. However, he was admitted three days later complaining of pain in his back and left shoulder. A tender mass was palpable in the left upper quadrant. His vital signs were stable. He subsequently underwent splenectomy.

Liver injury occurred in 15 patients. All had injuries to other areas of the body; rib fracture (seven patients) and head injuries (five patients) were the most common associated injuries. In four patients repair of the liver injury was delayed by more than eight hours. In each case the abdominal trauma had been masked by the associated injury. Delay in treatment was the leading factor in the death of one patient.

The pancreas was infrequently involved and was always associated with other abdominal injuries. There were no deaths due to pancreatic injury but the morbidity rate was high: one patient developed chronic relapsing pancreatitis and another a pseudocyst, which required internal drainage.

The bladder was the most frequently injured hollow viscus. Eleven of the 16 patients with such injury required operation because of perforation. All were treated successfully with suture repair, suprapubic cystostomy and urethral drainage. However, there was a high morbidity rate: eight of the 11 (73%) developed wound infection, cystitis or both.

Trauma to the bowel occurred in 14 patients. Definitive teatment was delayed by more than eight hours in five patients. Intra-abdominal injury was suspected but could not be proved. Physical findings were lacking and radiologic examination of the abdomen was negative. All five patients did poorly. One died of peritonitis and septicemia; the other four had prolonged morbidity because of peritonitis and wound infection. Of the eight patients that were operated upon within eight hours, there were no deaths and only one developed a wound infection.

Miscellaneous organs and supporting structures were damaged infrequently but presented interesting problems. One patient died from a laceration of the external iliac artery and vein. Another died from a com-

plete rupture of the right lobe of the liver associated with a tear of the portal vein.

Diagnosis and Discussion

The physical findings of peritoneal irritation, especially if progressive, are considered to be pathognomonic of intra-abdominal catastrophe. These findings include abdominal pain, rebound and rectal tenderness, absence of bowel sounds and distension. However, these findings are frequently either not present or are masked by injuries to other parts of the body. The following two cases illustrate this point:

Case 1.—F.W., a 58-year-old man, was involved in a car accident and was admitted to hospital in coma. Examination showed that he had sustained a fractured skull and a comminuted fracture of his left femur. Abdominal examination was reported as negative. Over the next 48 hr. his condition remained stable. However, on the third day he suddenly died. At autopsy he was found to have a massive hemoperitoneum from a ruptured spleen.

Case 2.—A.S., a 62-year-old man, was admitted because of multiple fractured ribs on the right side with associated hemopneumothorax. He complained of some abdominal pain and tenderness especially on the right side. There was moderate abdominal distension; the bowel sounds were present. The possibility of liver trauma was discussed but it was felt that the abdominal findings were probably secondary to the injury to the thorax. Over the next 10 days his condition slowly improved and the abdominal tenderness and distension decreased slightly. However, on the tenth day his condition suddenly deteriorated. Abdominal tenderness and distension became severe, bowel sounds disappeared. At laparotomy he was found to have laceration of the right lobe of the liver with blood and bile in the peritoneal cavity, and evidence of peritonitis. He died four days later of septicemia.

While these two cases illustrate very clearly the importance of early treatment, they also point out how difficult it can be to make an early diagnosis if associated injuries are present. Several authors[4, 9] point out that a correct diagnosis can be made only if there is a suspicion that an intra-abdominal injury may exist and if frequent and thorough clinical evaluations are carried out by the same clinician.

Although peritoneal aspiration was used infrequently in this series, it can be of great assistance in confirming a diagnosis. If performed in all four quadrants of the abdomen, the test is reported to be positive in 80% of cases.[4, 10, 11] A negative aspiration, however, is of no significance.

Elevation of pulse and temperature are of little value. A leukocytosis of above 20,000/c.mm. is reported to indicate injury to solid viscera.[9, 12] We could not demonstrate this relationship.

Blood in the urine is almost invariably present with injury to the urinary tract and radiologic examinations proved to be valuable in localizing the site of injury. However, radiologic examination proved to be of little assistance in making the diagnosis of perforated hollow viscus. Whereas the presence of free air is indicative of perforation, the absence of free air is of no value. Only two of 10 cases (20%) with perforated viscus had evidence of free air on radiologic examination.

The presence of fractured ribs on the left side is frequently suggested as being important when considering the diagnosis of ruptured spleen.[9] This relationship can be dangerous for, whereas, the presence of rib fracture must make one suspect the possibility of a ruptured spleen, the absence of a rib fracture should not make one any less suspicious. This is illustrated by the fact that over 70% of our patients with ruptured spleen did not have associated rib fracture. On the other hand, there seems to be a direct relationship between fractures of the bony pelvis and injury to the urinary bladder or urethra. Fourteen of 15 patients (93%) with contused or ruptured bladder had fractures of the pelvis.

It has already been pointed out that, when there are associated injuries to intra-abdominal organs, the signs and symptoms of peritoneal irritation are frequently absent. Abdominal aspiration, laboratory investigation and radiologic examinations can be of assistance in making a diagnosis but all these procedures have their limitations and are of little value if the results are negative. Thorough and repeated clinical evaluations of the patient seem to be, therefore, the best and surest method of making a positive diagnosis. However, if lapar-

otomy is not performed until a positive diagnosis is available, valuable time may be lost in the interim because, under certain circumstances, a diagnosis may be impossible for many hours or even days.

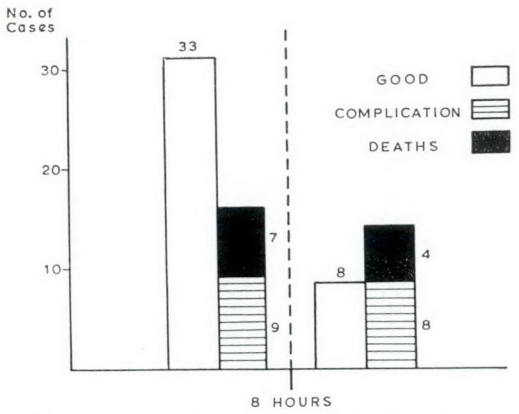

Fig. 3.—Graph comparing results of treatment in those patients who underwent laparotomy within eight hours of injury with those who had laparotomy after eight hours.

Figure 3 represents an analysis of results of treatment in patients who required laparotomy because of intra-abdominal injury. It is immediately apparent that results were far better when laparotomy was performed within eight hours of injury; 68% of those patients did well; whereas when laparotomy was delayed by more than eight hours only 40% did well.

We must therefore ask the question: is it essential to make a definitive diagnosis of intra-abdominal injury with peritoneal irritation before laparotomy is performed? We think not. We believe that laparotomy should be performed early (within eight hours) whenever possible and should be performed if there is even a *suspicion* of intra-abdominal injury. Surgical exploration is often the only means of making an early diagnosis. It is frequently impossible to determine the full extent of injury other than by surgical exploration. In patients subjected to laparotomy and found to have no intra-abdominal injury, Fitzgerald, Crawford and De Bakey[13] had no deaths, but had three deaths from intra-abdominal hemorrhage where exploration was not performed because the abdominal injury was masked by associated head injuries. In the present series four patients underwent negative laparotomy. There were no operative deaths. On the other hand, of the 20 patients in whom laparotomy was performed after eight hours, two died as a direct result of a delay of treatment.

Summary

A review of 110 cases of blunt abdominal trauma was presented. The mortality rate was 10%.

The difficulties of making an early diagnosis are discussed. It is pointed out that when there are associated injuries to extra-abdominal organs, the signs and symptoms of intra-abdominal injury are frequently absent, making early diagnosis virtually impossible.

A plea is made by the authors for laparotomy within eight hours of injury when there is even a *suspicion* of injury to intra-abdominal organs.

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Résumé

La mortalité résultant de lésions abdominales contondantes est plus forte que celle qui suit des blessures causées par un instrument qui a pénétré dans l'abdomen. Ceci est principalement la conséquence directe de la facilité du diagnostic initial dans le second cas.

On a étudié ainsi 110 malades qui présentaient des lésions intra-abdominales notoires. On attire l'attention sur les points suivants: (1) Ces blessures surviennent avec une plus grande fréquence chez les sujets compris dans le groupe d'âge de 10 à 30 ans, mais la plus forte mortalité s'observe aux deux échelons extrêmes de la vie. (2) Quand coexistent des lésions intra-abdominales et des blessures intéressant des organes extra-abdominaux, les signes et symptômes d'irritation péritonéale sont souvent absents, rendant difficile, voire impossible, un diagnostic précoce. (3) L'aspiration abdominale, les analyses de laboratoire et les examens radiologiques peuvent aider à porter un diagnostic, mais ces examens ont tous leurs limitations propres et n'ont guère de valeur si les résultats sont négatifs. (4) Le moyen le plus précis d'obtenir un diagnostic est de procéder à un examen du malade, à intervalles rapprochés. Même alors un diagnostic positif peut être impossible à porter avant plusieurs heures. (5) Une laparotomie faite dans un délai de huit heures même si l'exploration est négative, a pour effet de réduire la morbidité et la mortalité.

BREAST BIOPSY AS AN OUTPATIENT PROCEDURE

It is stated in most standard surgical texts and the opinion is held by most authorities in the field that biopsy of the breast should not be done under local anesthesia in the office or as an outpatient procedure. At first reading, the article in this issue by Doctor Abramson from the Walter Reed Army Medical Center would seem to be at variance with this well established surgical principle. Careful reading of Doctor Abramson's contribution would indicate that this is not the case.

Whenever a lesion appears suspicious of cancer the Walter Reed group have avoided outpatient biopsy and referred the patient into the hospital. Their study is a remarkable documentation of their clinical abilities in recognizing the early signs of cancer. When only lesions which they believed clearly to be benign were biopsied, the incidence of unsuspected cancer in the 857 biopsies was just under 5%. This group of 41 patients who were promptly admitted to the hospital and had a radical mastectomy performed within 72 hours or less of the biopsy seem not to have suffered any ill effects as far as a statistical analysis of the results would indicate. The overall five-year survival rate was 78.4%. Patients without axillary metastases had a 90% five-year arrest and those with axillary metastases a 44% five-year survival rate. It should be recognized, of course, that these represented patients with such early cancers that they were initially diagnosed as being benign lesions.

The most important feature of the study is the combination of rigid selection of patients for biopsy on an outpatient basis, immediately available highly skilled pathologic examination by frozen section, and such control of both patient and hospital as to permit immediate arrangements for admission and radical mastectomy within two or three days at the most. It must also be remembered that the lesions were small and total excision biopsy was practised.

It is unlikely that the exact circumstances available to these workers can be found in private practice. Moreover, most community and university hospitals are not geared for this type of patient care. Centres concentrating primarily on carcinoma of the breast might, in the same selected cases, adopt a similar procedure. There is no doubt that with large numbers of patients presenting for biopsy of the breast this approach can afford a remarkable saving in hospitalization and total costs. On the other hand, one cannot escape the impression that there were probably many patients in this series that would not have been subjected to either biopsy or hospitalization by experienced surgeons in private practice. Particularly in the young, a small nodule in the breast may be observed safely for a week or two and found to regress with the next menstrual period.

The availability of the staff and facilities for this type of study and the pressure placed upon the surgeon by the anxious patient with a palpable nodule would seem very likely to have weighted the series and been another factor in the low incidence of proved carcinoma.

It would seem obvious that the indications and technic for biopsy of the breast in patients with suspected carcinoma would maintain its traditional place in the hands of individual surgeons, namely, biopsy under circumstances which permit frozen section and radical mastectomy in the same sitting.—Dunphy, J. E.: Editorial. Breast biopsy—indications and technic, *Ann. Surg.*, **163:** 484, 1966.

REVIEW ARTICLE

BLUNT ABDOMINAL TRAUMA*

R. A. MACBETH, M.D., F.R.C.S.[C],† *Edmonton, Alta.*

THE diagnosis and management of blunt abdominal injuries are probably among the most perplexing problems that face the surgeon. While not exclusively the result of motor vehicle accidents, such was the etiology in fully 62% of our series of patients, and the slaughter on Canadian highways is increasing year by year.

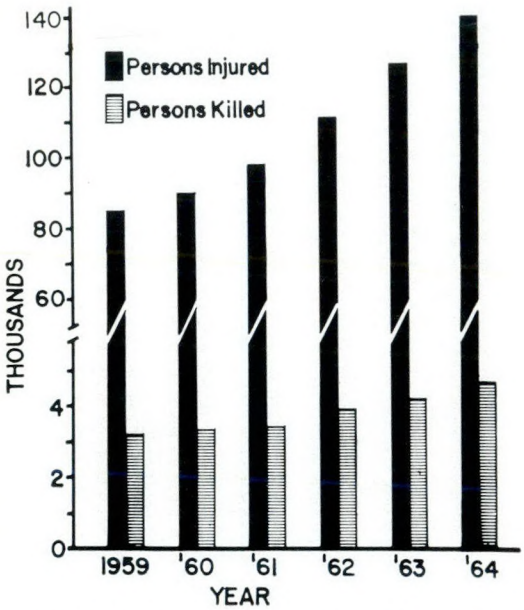

Fig. 1.—Traffic accidents in Canada (Canadian Highway Safety Council).

Figure 1 summarizes statistics compiled by the Canadian Highway Safety Council.[1] Noteworthy are the 139,534 persons injured and the 4655 persons killed during 1964, the most recent year for which complete returns are available. These figures represent increases of 11.5% and 10.6% respectively over the 1963 figures, during which time our population increase amounted to 1.8%.

Root and Christensen[2] have carefully studied 1988 consecutive traffic victims treated in their hospital in Oakland, California. Abdominal injuries occurred in only 44 patients (2.2%) of the entire series but in this small group the mortality rate was 45%. Viewed another way, there were 45 deaths in their series and of these 20 (44%) were associated with abdominal injuries, either alone or in combination with injuries to other organ systems. While the comparison may not be valid, it would suggest that in Canada in 1964 some 3000 persons probably received abdominal injuries as a result of motor vehicle accidents and that probably an abdominal injury was the sole or a contributing cause of death in some 1500 instances. Whether one is prepared to accept this projection or not, there is little doubt that patients suffering from blunt abdominal trauma present a challenge to the medical profession of ever-increasing magnitude and that, although prevention rather than treatment must be our objective, familiarity with the diagnostic features and management of these lesions is mandatory if we are to reduce the carnage on Canadian highways.

For this reason we were stimulated to review our experience at the University of Alberta Hospital, Edmonton, over the 14-year period up to and including 1964. Criteria for inclusion of patients in the present study were as follows: (1) Only patients who were admitted to hospital were included. Patients dead on arrival at hospital or dying in the emergency department before definitive therapy could be initiated were excluded. (2) Only patients in whom the diagnosis was confirmed, either by operation or autopsy, were included. Excluded, therefore, were innumerable patients who were discharged with diagnoses of contusions of the abdominal wall, retroperitoneal hematomata or suspected injury to an intra-abdominal viscus, but who were managed without operation. (3) Patients with traumatic lesions of the urinary tract were excluded.

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†Department of Surgery, University of Alberta Hospital, Edmonton, Alta.

TABLE I.—ABDOMINAL INJURIES—UNIVERSITY OF ALBERTA HOSPITAL, EDMONTON, 1951 - 1964

Penetrating abdominal injuries		13
Blunt abdominal injuries		100
Splenic injury	53	
Hepatic injury	17	
Combined splenic and hepatic injury	9	
Gastrointestinal perforation	10	
Pancreatic injury	5	
Mesenteric laceration	4	
Retroperitoneal hematoma	2	

The classification of the 100 patients who fulfilled these criteria is shown in Table I.

During the same period, only 13 patients suffering from penetrating abdominal injuries were treated and, of these, the injuries in all but two were accidentally inflicted. In this regard, our experience tends to parallel that of the United Kingdom and the Scandinavian countries[3-5] where abdominal injuries are relatively infrequent and predominantly closed. Experience in certain areas of the United States appears to be in sharp contrast to this and numerous series attest to the frequency of abdominal wounding in metropolitan American cities and to the pre-eminence of penetrating injury among these wounds. In Texas, for example, approximately 90% of hepatic injuries were the result of penetrating trauma.[6, 7]

In this paper, when injuries due to blunt abdominal trauma are discussed and it is stated that the spleen alone, or the liver alone, was injured, this means that this organ *alone* of the intra-abdominal viscera excluding the urinary tract, was involved. Concommitant injury to the urinary tract or to extra-abdominal organ systems was, however, a relatively common occurrence.

Certain points in the history of patients with suspected abdominal injury emerged from this study: (1) The site of impact on the body may give a clue to the underlying pathology. While precise information in this regard was not often recorded on the hospital charts reviewed for this study, it was invariably true that, in those cases in which the site of impact was known, the underlying pathology was localized to that quadrant of the abdomen. (2) The severity of the trauma also appeared to bear some relationship to the injury sustained. Fully 85% of the 26 hepatic, and combined hepatic and splenic injuries were due to motor vehicle accidents and the trauma sustained by the remaining four patients was usually of comparable severity. Only 50% of the splenic ruptures were due to automobile accidents and in some of the remaining patients the injury that produced splenic rupture appeared to be minimal indeed. While only 30% of the gastrointestinal and 55% of the miscellaneous injuries were due to automobile accidents, the injury in the remaining patients in these groups was usually such as to be classed as relatively severe trauma. (3) The general history also proved to be most helpful in alerting one to the possibility of injuries to other organ systems. It is of extreme importance that patients with a multiplicity of injuries be recognized since fatalities almost invariably fall within this group, for example: If one organ is injured, there is a 15% mortality; if two organs are injured, there is a 28% mortality; if three organs are injured, a 46% mortality; if four organs are injured, a 62% mortality; and with five organs injured, 93% mortality.

SPLENIC INJURY

Certain features of specific organ injury become apparent on review of this group of patients, which appear worthy of mention. The 53 patients who sustained isolated splenic rupture (as their *intra-abdominal* injury) provided the largest group with blunt abdominal trauma.

Etiology

The mechanism of injury relative to age (Fig. 2) is of particular interest. The sex ratio was four males to one female. Note that in the first two decades, falls were the commonest cause of injury and this was particularly true of teen-agers. They fell out of trees, off fences, horses and roofs, and in fact almost anything else that one can name that it is possible to climb. From age 21 on, falls were a much less frequent etiological factor, and when they did occur they were usually the result of an industrial accident.

That portion of the graph that records accidents involving occupants of a car demonstrates that this mechanism is the major contributor to splenic rupture in the

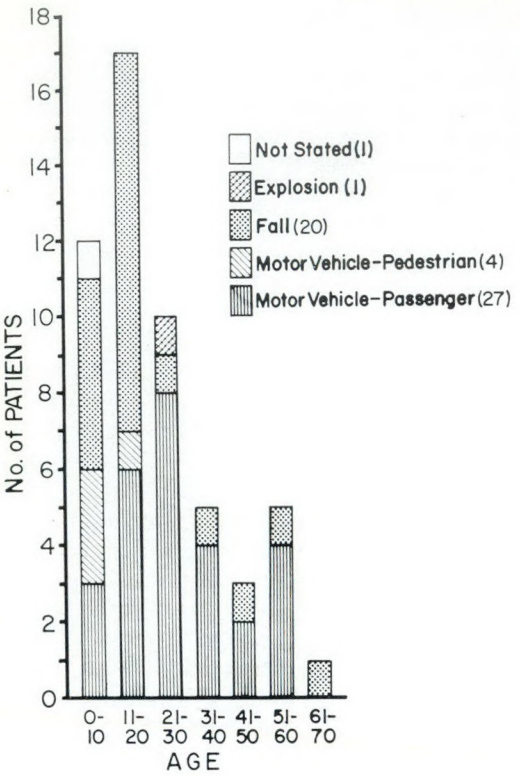

Fig. 2.—Splenic injury in 53 patients relative to age.

third decade and that it tapers off in the younger and older age groups.

In Fig. 2 it will be noted that the pedestrian is not immune to this injury. In the under-10-year group, this mechanism contributed as many pedestrian-patients as were observed among children who were passengers within the car. The teen-ager, being more nimble, is apparently seldom run down, while after 21, with a caution born of maturity, the pedestrian has apparently learned to avoid splenic injury in his struggle for survival.

It is worthy of note that 60% of the splenic ruptures occurred in the first two decades. The increased incidence of this lesion in the younger age group is well documented in the literature.[8]

Symptomatology

The symptomatology in 20 patients with splenic injury was unavailable in the charts. In five instances the patient was unconscious; in an additional 15 patients, either because of age, lack of co-operation on the part of the patient, or negligence on the part of the interrogator, the recording of the history was grossly inadequate.

In the remaining 33 patients, the complaint of pain restricted to or maximal in the left upper quadrant recurred with monotonous regularity. In 10 patients the pain was described as abdominal without further localization. Two patients, interestingly, denied the presence of any abdominal pain. In one, pain was restricted to the left shoulder and in one, it occurred only in the back.

It was of particular interest to note that left shoulder-tip pain was specifically complained of by 20 of the patients. Kehr's sign* would therefore appear to have been a useful diagnostic observation in about two-thirds of the patients in whom a satisfactory history was obtainable.

The infrequency of the complaint of weakness or fainting appeared somewhat surprising. It was recorded in only seven of the charts. Vomiting occurred in only three patients.

Physical Findings

While the record of the interrogation of these patients, many of whom were in acute distress, was often inadequate, the physical examination was usually recorded in detail.

We had anticipated that virtually all patients with splenic rupture would manifest a shock-like state on arrival at hospital. Such was not the case. The admission systolic blood pressure of 63% of the patients exceeded 100 mm.Hg, while 43% had a pulse rate below 100/min. The absence of shock on admission obviously does not exclude splenic injury. Boley, McKinnon and Schwartz[9] have made a point of emphasizing the frequent absence of the systemic manifestations of hemorrhage in children with traumatic splenic rupture.

In most of the patients, the findings on abdominal examination were classical (Table II). Usually tenderness was present only in, or was clearly maximal in, the left upper quadrant. Noteworthy, however, are the five patients in whom tenderness was restricted to the right upper quadrant. In three of these patients, muscle guarding

*Left shoulder-tip pain frequently intensified with respiration.

TABLE II.—Splenic Trauma. Physical Findings

Abdominal tenderness:	
Left upper quadrant	27
Generalized	6
Right upper quadrant	5
Absent	3
Back only	1
Guarding:	
Left upper quadrant	16
Generalized	9
Right upper quadrant	3
Rebound tenderness:	
Present	10
Absent	8
Bowel sounds:	
Normal	12
Reduced	9
Absent	12

was present, had the same localization and resulted in an erroneous preoperative diagnosis of liver injury and the performance of a right paramedian incision. Careful laparotomy failed to reveal any lesion of the liver but, in each, the spleen was badly lacerated necessitating extension of the incision to the left to permit splenectomy. Retrospective chart review in these patients revealed that in three of the five, the site of the trauma was recorded to be in the left upper quadrant and it would have been a more valuable guide to the organ injury than were the abdominal findings.

Abdominal muscle guarding was less commonly present than was tenderness but tended also to be maximal in or restricted to the left upper quadrant. In degree it tended to be moderate as compared with that observed following peritoneal contamination by gastrointestinal contents. Rebound tenderness was present in about half of the patients in whom it was sought after and recorded, but was usually mild in degree. The bowel sounds were present in 12 patients and diminished or absent in 21. The frequency of these latter two findings is to be compared with that observed in gastrointestinal-tract perforation.

Ballance's sign* was apparently not sought after, or at least was not recorded on any of the charts. Shifting dullness was, however, noted to be present twice in the four patients examined for its presence.

Delayed Splenic Rupture

Nine (17%) of the 53 patients with splenic injury had a clinical course that fulfilled the requirements for classification as delayed rupture of the spleen. All experienced a period of complete freedom from symptoms which varied from five to 14 days following injury. This interval was followed by the apparently spontaneous development of the catastrophic signs of massive hemoperitoneum. In three patients the initial injury had not been considered of sufficient magnitude by the patient that medical attention was sought, or if sought, was considered of little significance by the attending doctor. Six of the patients were admitted to hospital following the accident, either because of associated injuries or because of the suspicion that an intra-abdominal lesion existed. However, abdominal findings, if present, rapidly disappeared and three of the hospitalized patients had, in fact, been discharged home before the delayed rupture of their spleen. The drama inherent in the story related by these patients obviously caught the fancy of the attending houseman for their history was invariably recorded in vivid terms. In one patient the history of trauma was not elicited until after laparotomy had demonstrated the splenic rupture. This 25-year-old nurse, who collapsed while at work, manifested signs of hemoperitoneum and was considered to have a ruptured ectopic pregnancy for which laparotomy was expeditiously performed. Only in the post-operative period, when the diagnosis of splenic rupture had been established, was the history obtained from the patient that five days previously she had slipped and fallen on an icy street and sustained what she considered to be an insignificant bruise of her left lower chest.

Splenic Rupture Associated with a Head Injury and Unconsciousness

Of particular interest were a group of five patients with splenic rupture who were unconscious on admission because of an associated head injury. In these patients the credit for initiating appropriate and life-saving surgical intervention must be given to the attending neurosurgeon. Review of these charts is most interesting. In the first scene one can almost see the neurosurgeon insisting that the shock-like state that has

*Fixed dullness in the left flank, with shifting dullness in the right flank.

developed cannot possibly be due to the head injury and that blood loss must be occurring in some region remote from the head. On the other hand, one recognizes the reluctance of the consultant general surgeon to perform a laparotomy, in the absence of clear-cut abdominal findings, in a deteriorating, unconscious patient. Invariably he notes that there is no sign of intrathoracic bleeding, no source of significant blood loss in the extremities and that, if continued hemorrhage is, in fact, taking place it must be intra-abdominal and probably arising from the spleen. The second scene fortunately takes place in the operating room with the still-reluctant general surgeon preparing for laparotomy. The climax comes as the discoloured peritoneum is opened revealing the hemoperitoneum and the splenic laceration. The moral of this little drama is obvious. A head injury in itself does not cause shock and, in the presence of shock in such a patient, the source of continued bleeding must be sought after, by exclusion if necessary, and appropriate therapy initiated at once.

Wilson, Vidrine and Rives[10] have drawn attention to the difficulties in diagnosis, and the increased mortality associated with unrecognized abdominal trauma in patients with head injuries. From a mortality of 15% in patients with abdominal injury only, the mortality rose to 76% in those in whom an abdominal injury was associated with coma due to a head injury. They recommended "four-quadrant" diagnostic abdominal tap in such patients and reported a 95% accuracy with this procedure. While we would agree with their recommendation, our own experience with diagnostic abdominal tap has not been as rewarding. It was seldom performed in this series of patients with proved intra-abdominal pathology and was negative as often as it was positive. However, it was not possible to be sure that all four quadrants were routinely tapped in our patients and the validity of our unsatisfactory experience with this procedure is open to question.

Laboratory Findings

The admission laboratory findings in this series of patients is of some interest and

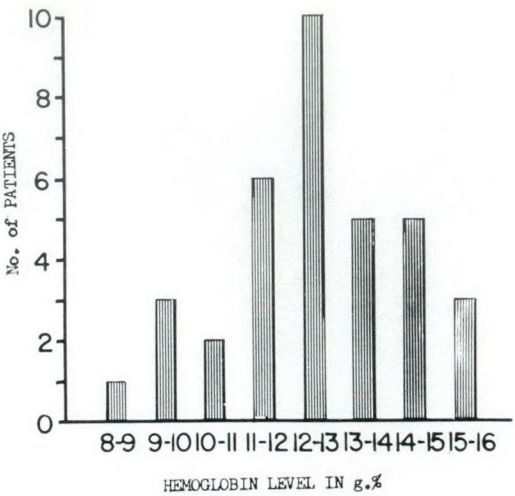

Fig. 3.—Admission hemoglobin level in 53 patients with splenic trauma.

importance. Fully 65% of those patients in whom a preoperative hemoglobin estimation was available had values in excess of 12 g.% (Fig. 3). Therefore, while serial hemoglobin determinations may be useful in questionable cases of blood loss, a single admission determination appears to have little diagnostic value.

The leukocyte count, on the other hand, appeared to be of value in predicting the presence of intraperitoneal blood (Fig. 4).

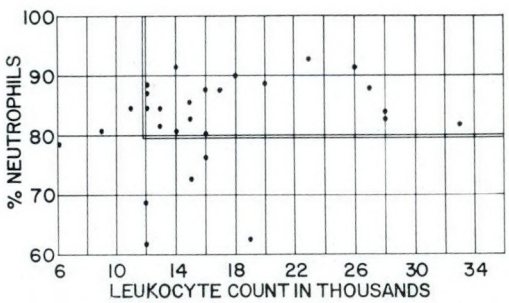

Fig. 4.—Admission leukocyte count in 28 patients with splenic trauma.

Fully 72% of the patients in whom preoperative leukocyte counts were recorded, manifested total counts in excess of 12,000/c.mm. with a polymorphonuclear leukocytosis in excess of 80%. These findings would support the opinion expressed in the literature[8, 9, 11] that: (1) blood in the peritoneal cavity of hepatic or splenic origin evokes an even greater polymorphonuclear leukocytosis than do gastrointestinal con-

tents, and that, (2) the leukocyte count is more helpful in suggesting the presence of a ruptured spleen than is the hemoglobin determination.

However, few would disagree that the diagnostic value of a careful clinical assessment of the patient far outweighs the value of any known laboratory procedure.

Associated Injuries

The proved associated injuries encountered in the 49 patients who survived splenic trauma are summarized in Table III. The skeletal system was the most common site of associated injuries. In all probability more than 12, or one-quarter, of these patients sustained fractures of the left rib cage, but in this number at least radiological confirmation was available. As may be seen, virtually all other types of fracture were occasionally recorded as an associated injury. Intrathoracic injuries ranked second in order of occurrence, followed by cerebral and renal lesions. In the surviving patients, all of the renal lesions were managed without operation, but, of the four patients who died, two required nephrectomy because the left kidney was pulverized.

TABLE III.—Splenic Trauma. Associated Injuries (49 Surviving Patients)

Fractures		28
Ribs	12	
Lower extremity	4	
Upper extremity	3	
Vertebrae or transverse processes	4	
Pubis	3	
Skull	2	
Intrathoracic		7
Hemothorax	5	
Pneumothorax	2	
Cerebral contusion (with unconsciousness)		5
Associated hemiparesis	2	
Renal contusion		3

Hepatic Injuries

In patients with hepatic injury, or combined hepatic and splenic injury, the history was almost invariably one of severe trauma. Fully 22 of the 26 patients were involved in motor vehicle accidents either as occupants of the car or as pedestrians. Of the four remaining patients, all of whom suffered isolated liver injury as the intra-abdominal component of their traumatic insult, one was crushed under a house that was being moved, one girl, aged 14, was thrown from a horse, and one workman, aged 38, was drawn into an industrial machine when his clothing became entangled with the crankshaft. All therefore, except one child to be referred to later, suffered a severe traumatic insult.

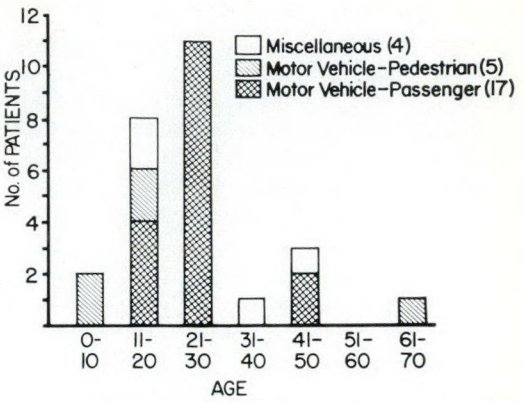

Fig. 5.—Hepatic injury in 26 patients related to age.

The predominance of the male is again apparent (Fig. 5). Unlike the group of patients with splenic injury, the maximum age incidence in this small group of hepatic and combined lesions occurred in the third decade and such injury was relatively uncommon in children under 10 years of age. The paucity of this lesion in the latter age group is, however, not a feature of all published series.[3]

In patients with liver injuries, severe injuries of other organ systems often coexisted, confused the clinical picture and contributed to the increased morbidity and mortality. Shock was much more commonly present on admission in these patients than in patients with splenic injury and frequently it was of profound degree. The symptoms and signs, where these were obtainable, were essentially similar to those occurring in patients with splenic rupture except that they were localized to, or were maximal in, the right upper quadrant.

Three patients with liver injury were of sufficient clinical interest to warrant brief individual mention.

Post-traumatic Bile Peritonitis

The course of two patients followed a remarkably similar pattern. Both were involved in catastrophic motor vehicle acci-

dents and were admitted unconscious with injuries to multiple organ systems. In both, the presence of abdominal findings on initial examination were not considered sufficient to warrant laparotomy in these seriously ill patients. However, jaundice subsequently became clinically manifest, on the fourth day following trauma in one patient and on the fifth day in the other, and was associated with progressive abdominal distension and paralytic ileus, although other abdominal findings were minimal. At laparotomy, in one patient six and in the other 14 days following injury, they were found to have 5500 and 6400 c.c. respectively of serous, bile-coloured fluid within the peritoneal cavity and each had a liver laceration that was not actively bleeding. Simple drainage of the peritoneal cavity was followed by an uninterrupted recovery. The benign nature of bile peritonitis following liver injury is in sharp contrast to that observed following perforation of an inflamed gallbladder.

Intrahepatic Hematoma

The other patient of interest was a 12-year-old Metis girl who, while running, tripped and fell striking the right upper quadrant of her abdomen on a water pail. She represented the only case of liver injury unassociated with what was obviously a major traumatic insult. After observation in a northern hospital, she came to laparotomy at the University Hospital, Edmonton, 19 days following her accident because of persistent fever and a large right upper quadrant mass. She proved to have an encysted liquifying hematoma of the liver containing 1500 c.c. of old blood. Drainage of the cyst by means of a mushroom catheter proved curative.

Gastrointestinal Perforation

Among patients with gastrointestinal perforation due to blunt abdominal trauma, motor vehicle accidents were much less frequently the etiological factor than was the case with liver injuries, although the magnitude of the trauma was usually of comparable severity. Unlike solid organ injury, in which the site of impact was invariably upper abdominal, gastrointestinal injuries occurred following trauma to any one of the abdominal quadrants.

One feature of the pain in traumatic intestinal perforation was that, whether it came on suddenly or gradually, it tended to increase in severity and become generalized in almost all cases. This was in contradistinction to the pain manifested by many of the patients with solid-organ injury in whom the pain did not tend to progress in severity or become generalized.

The site of the intestinal perforation in these 10 patients is indicated in Table IV.

TABLE IV.—Site of Intestinal Perforations in 10 Patients

Duodenum		1
Small bowel		8
Jejunum	5 (2 multiple)	
Ileum	2 (2 multiple)	
Unstated	1 (1 multiple)	
Colon (also jejunal)		1
Rectum		1
Associated bowel contusions	2	
Associated traumatic pancreatitis	1	

The duodenal perforation occurred as an isolated injury in a two-year-old child and proved to be lethal. The eight small-bowel perforations tended to occur in the more fixed portions of the intestine, five occurring in the proximal jejunum and two in the terminal ileum. In five instances, multiple small-bowel perforations occurred. The transverse colon was perforated at its midpoint in a patient who also had a jejunal perforation, which accounts for the 11 tabulated sites of bowel perforation in the 10 patients. The final patient suffered a rectal perforation.

Insofar as the signs present in patients with hollow-viscus perforation were concerned, abdominal tenderness was present in nine patients on admission and in all by the time of operation. The tenderness elicited was exquisite, to a degree not commonly seen with solid organ injury. In the child with duodenal perforation, it was subsequently presumed that the initial period associated with absence of tenderness corresponded to the time of retroperitoneal perforation, and that tenderness subsequently occurred with peritoneal dehiscence and intraperitoneal contamination. Rebound tenderness was likewise more common and intense in gastrointestinal perforation, occurring in half of the patients.

Board-like rigidity was present in six patients with definite muscle guarding in the remaining four. Board-like rigidity was not a feature of solid-viscus injury and, although some degree of muscle guarding was commonly present, the abdomen was frequently described as offering resistance rather than manifesting true guarding or rigidity. In the seven patients in whom bowel sounds were sought, they were absent.

Pancreatic Injuries

The five patients with pancreatic lesions fell into two distinct clinical groups. Two patients were subjected to operation shortly after injury because of progressive signs of peritoneal irritation associated with marked elevation of serum amylase levels. In both,

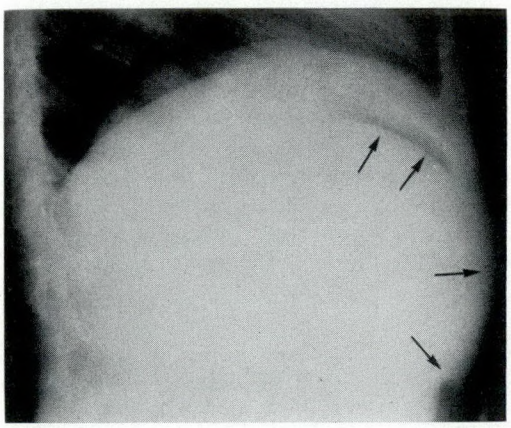

Fig. 6.—Post-traumatic pancreatic pseudocyst. Preoperative lateral erect scout film of the abdomen.

drainage to the exterior by means of Penrose drains was all that was deemed possible in the presence of extensive contusion, maceration, and edema of the gland. Convalescence was prolonged and associated with protracted drainage, but recovery was eventually complete.

In the three remaining patients with pancreatic lesions, abdominal injury had occurred 10 days to two weeks previously with persistence of upper abdominal symptoms and signs necessitating transfer to a city hospital. All manifested an epigastric mass on admission. Figures 6 to 10 are photographs of representative radiographs taken on one of these patients, which are illustrative of the roentgenographic findings present in all. Note the gastric air bubble displaced anteriorly by the globular retrogastric mass in the lateral scout film of the abdomen (Fig. 6). Meglumine diatrizoate (Gastrografin) swallow clearly shows the

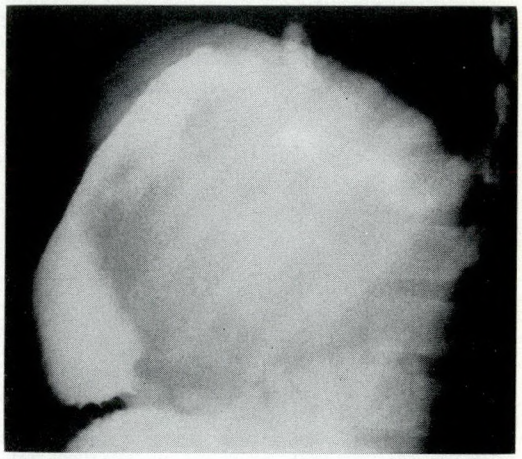

Fig. 7.—Post-traumatic pancreatic pseudocyst. Preoperative lateral roentgenogram following ingestion of Gastrografin.

same anterior displacement of the stomach in the lateral view (Fig. 7) while, on the spot films of the anteroposterior projection,

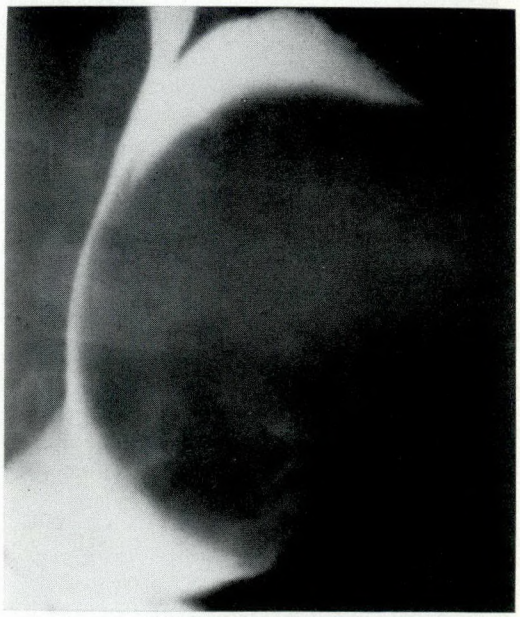

Fig. 8.—Post-traumatic pancreatic pseudocyst. Preoperative anteroposterior spot film of the stomach following ingestion of Gastrografin.

one sees the rim of opaque medium on the lesser curvature and, to some extent, the greater curvature margins of the stomach while its central portion, displaced anteriorly and compressed by the retrogastric mass, fails to fill (Fig. 8). Four weeks after operation, the Gastrografin-filled stomach is seen to have returned to its normal position in the lateral (Fig. 9) and the antero-

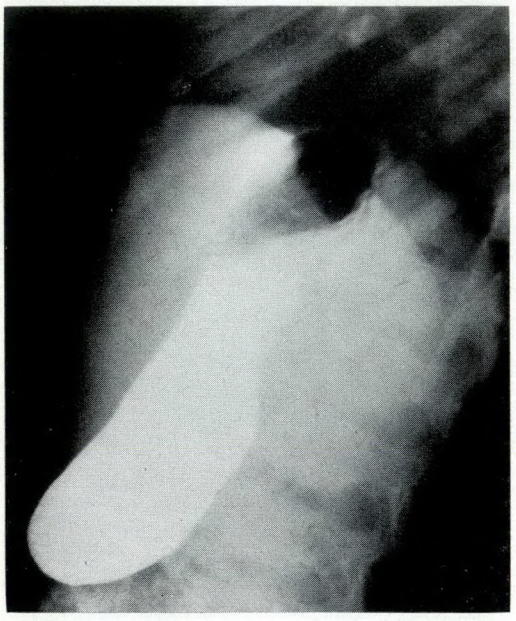

Fig. 9.—Post-traumatic pancreatic pseudocyst. Postoperative (four weeks) lateral roentgenogram following ingestion of Gastrografin.

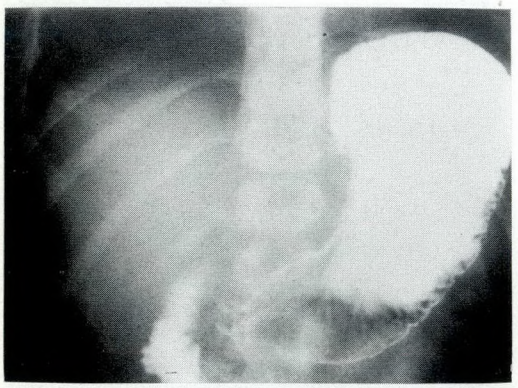

Fig. 10.—Post-traumatic pancreatic pseudocyst. Postoperative (four weeks) anteroposterior roentgenogram following ingestion of Gastrografin.

posterior projections (Fig. 10). No Gastrografin is seen entering the cyst.

In all three patients, cystogastrostomy was performed for a typical post-traumatic pseudocyst of the pancreas. All patients survived and were discharged symptom free, although hospitalization was prolonged.

Miscellaneous Injuries

The remaining six patients require little comment. The two patients with retroperitoneal hematomata were included because acute abdominal symptoms and signs led to laparotomy. During the same period many additional patients were discharged with the same diagnosis but in these the symptoms subsided without laparotomy and, since the diagnosis was not confirmed, they have not been included. Of the two patients explored, one was drained and one was not; both made an uninterrupted recovery. Two subsequent operations for intestinal obstruction due to adhesions were, however, necessary at a later date in the patient who had the hematoma drained.

Finally, there were four patients with mesenteric tears, one involving the mesentery of the upper jejunum and three that of the terminal ileum. As with solid organ injury, the symptoms and signs were those of hemoperitoneum. In three patients, ligation of severed vessels and repair of the mesenteric laceration was accomplished without devitalization of bowel and convalescence was uneventful. In the fourth patient, extensive contusion and laceration of the mesentery of the terminal ileum was associated with devitalization of 18 in. of bowel, which required resection. Associated injuries of the skeletal system necessitated prolonged hospitalization but recovery from the abdominal operation was prompt.

Scout Roentgenograms of the Abdomen

In this total experience with blunt abdominal trauma, scout films of the abdomen were seldom of assistance in differential diagnosis. Occasionally an enlargement of the splenic shadow or an increased serration of the greater curvature of the stomach was noted in connection with rupture of the spleen. Occasionally too, a so-called "ground-glass" appearance suggested to the radiologist that free fluid was present in the

abdominal cavity. However, it appeared that in no case was the diagnosis dependent on the interpretation of the scout roentgenograms. Even in cases of traumatic perforation of the bowel, none of the patients in this series who were examined radiologically manifested subphrenic air in the upright film. This agrees with the findings of Cooke and Southwood[12] who observed subphrenic gas in most patients with rupture of the stomach or colon, but in only the occasional patient who had sustained rupture of the small bowel. It is to be noted that eight of our patients suffered small-bowel perforation.

General Management of Blunt Abdominal Injuries

In the management of patients with blunt injuries to the abdomen, resuscitation should proceed concurrently with history-taking, physical examination and other diagnostic procedures.

A reasonable order of priority in the management of the severely injured patient is as follows:

(1) Secure an adequate airway and deal with any life-endangering thoracic injury.
(2) Arrest obvious hemorrhage.
(3) Initiate a record of the vital signs.
(4) Draw blood for cross matching and insert a large-bore needle or plastic catheter into a good-sized vein. Depending on the circumstances this can be kept open with glucose and water or plasma expanders, such as dextran, until appropriate matched blood becomes available.
(5) Splint fractures.
(6) Insert a bladder catheter.
(7) Insert a Levin tube.
(8) Obtain scout films of the abdomen, intravenous pyelograms and cystograms, and such other radiographic studies as are indicated.
(9) Obtain such laboratory work as is indicated including, on occasion, a serum amylase.

It should, at this point, be possible to decide whether urgent surgery is indicated. It is usually possible to control shock by blood transfusion before surgery but sometimes shock persists due to blood loss in excess of replacement therapy and in these desperately ill patients urgent laparotomy, even in the face of progressive shock, is the only possible means of saving life.

Conduct of Laparotomy in Patients with Blunt Abdominal Trauma

In connection with laparotomy in patients with blunt abdominal trauma, a planned approach is of extreme importance.

(1) The incision should be adequate and capable of rapid extension. Probably a paramedian incision is ideal in this situation. In the present series of patients with splenic rupture, a left paramedian incision was used in 70%, and a subcostal or transverse incision in 18%.
(2) If hemorrhage is present, its control is obviously of prime importance. One should remember that the organs likely to give rise to this hemorrhage are, in order, the spleen, the liver, the mesenteries, the small bowel, the stomach and the kidneys.
(3) Once hemorrhage is controlled, or if no blood is present, observe any intra-abdominal fluid carefully for evidence of bile or gastrointestinal contents and then aspirate it. A culture of this fluid and its analysis for amylase may be helpful in the postoperative management.
(4) After examination of the mesentery of the small bowel for lacerations, the small bowel should be brought out and a careful search made throughout its entire length for evidence of injury.
(5) The stomach is then inspected, including its posterior wall.
(6) Attention is then directed to the colon and retroperitoneal structures, particularly the duodenum and the pancreas. The liver and spleen are finally examined if this has not already been done.

Management of Specific Organ Injury

Any trauma to the spleen is an indication for splenectomy. Drainage of the region of the pancreatic tail is usually stated to be desirable following splenectomy. Drainage was carried out in 72% of our patients with

splenic injury, without complication attributable to drainage. However, it should also be noted that no complication occurred in the 28% of patients in whom a drain was not used, which might be attributed to its absence. It is difficult to prove either that using drains or not using them was helpful. It is, however, true to say that putting in a drain did no harm but, on the other hand, there is no clear evidence that any trouble occurred because it was not used.

Liver lacerations should ideally be treated by suture or, if maceration of a portion of the liver has occurred, by resection of devitalized tissue or by lobectomy. Hepatic lobectomy was not carried out in this series although we agree that resection should be done in the patient with a severely lacerated and fragmented liver. It is of interest to note in the literature that, while resection is not infrequently recommended, few series describe any significant experience in its performance, except for the recent report of McClelland and Shires[7] in which 25 resections were done, 11 of which were right or left lobectomies.

Of the 13 patients with isolated liver injury in this series who survived operation and in whom the efficacy of the surgical treatment could be assessed, only one patient had a laceration situated in a sufficiently accessible portion to permit suture. In five patients, bleeding had either stopped by the time of laparotomy or was controlled with hot packs, which were subsequently removed. In these patients, drainage alone was carried out and no intra-abdominal complications ensued. In five patients, the laceration required packing with some absorbable hemostatic agent and in three of these infection occurred and prolonged the recovery phase. One of the remaining patients was treated by insertion of a mushroom catheter into a hematoma cavity while in the other the cavity was packed with vaginal packing. Both these latter patients made an uneventful recovery. The necessity of adequate drainage is well recognized[6, 7, 12-14] and this was provided in all our patients.

Packing of liver wounds with gauze packing is generally, and very reasonably, condemned[6, 12] since secondary hemorrhage on pack removal and infection so commonly follow this method of management. Absorbable hemostatic agents have received a much more favourable reception in the surgical literature, although our own experience suggests that a significant risk of infection is associated with the use of these agents. It appears, however, that where suturing proves to be impossible and the operator has had little experience with hepatic resection, packing, preferably with an absorbable hemostatic agent, may well offer the patient the best chance of survival in spite of the risk of suppuration.

Surgical decompression of the biliary tree has recently been advocated in extensive hepatic lacerations and injuries that extend deep into the liver parenchyma.[2] This procedure was not used in this series but it appears to be a useful adjunct to therapy.

Gastrointestinal injuries resulting from blunt trauma should be managed by different techniques at different levels: (1) Stomach perforations are best managed by minimal debridement and two-layer closure. (2) Small-bowel perforations lend themselves to minimal debridement and two-layer closure, or, if extensive, to treatment by resection and primary anastomosis. (3) Colonic injuries are most safely treated by exteriorization. Closure and proximal colostomy is not to be recommended, except in the case of rectal lacerations in which no other alternative exists.

Pancreatic injuries, if of the tail, are best treated by resection. In the body, while distal resection is probably still ideal and is apparently unassociated with subsequent pancreatic insufficiency,[15] simple adequate drainage may well prove lifesaving. Various ingenious methods of *Roux-en-Y* pancreaticojejunostomy have been described for transection of the body of the pancreas[16] but have a limited application since ideal conditions for the performance of these procedures seldom exist. Traumatic lesions of the head of the pancreas are almost invariably fatal and no satisfactory treatment exists. Traumatic pancreatic pseudocysts may very effectively be treated by cystogastrostomy (or *Roux-en-Y* cystojejunostomy) as illustrated in the three cases reported here.

Post-splenectomy Thrombocytosis

The postoperative care of patients treated for blunt abdominal trauma presents few special problems. The thrombocytosis that follows removal of a traumatized normal spleen is, however, of considerable interest. Figure 11 graphically

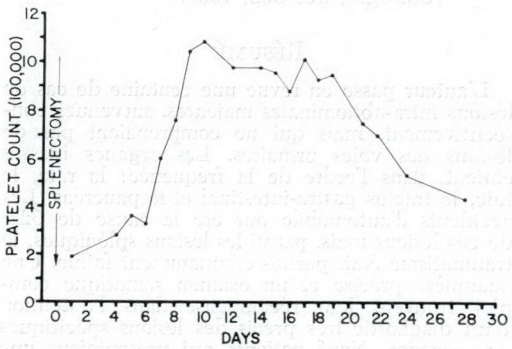

Fig. 11.—Thrombocyte response to removal of a traumatized, otherwise normal, spleen.

portrays the typical response seen in these patients. In 32 patients in this series in whom serial platelet counts were available, 25% had elevations in excess of one million/c.mm. while only 15% showed elevations less than 500,000/c.mm. Whether this thrombocytosis contributes to postoperative intravascular thrombosis is not universally accepted but certainly the possibility exists, and it has been the practice in our hospital for the last number of years to perform serial platelet counts on such splenectomized individuals and initiate anticoagulant therapy if platelet counts exceed one million/c.mm.

TABLE V.—Mortality Rates Associated with Specific Organ Injuries

Injury	*No. of patients*	*Mortality (%)*
Splenic	53	7.5
Hepatic	17	17.7
Splenic and hepatic	9	88.9
Gastrointestinal perforation	10	20.0
Miscellaneous	11	0.0

Mortality

The overall mortality in this series was 17%. The mortality figures for specific organ injuries are summarized in Table V.

Splenic rupture carried a mortality of 7.5%. However, three of the four deaths occurred in patients with grave injuries to multiple organ systems and could not be attributable to the splenic injury *per se*. Only one patient died who suffered splenic injury alone (of a pulmonary embolus on the seventeenth postoperative day).

In patients in whom liver injury constituted the only intra-abdominal injury, the mortality rate was 17.7% in spite of the fact that many of these patients suffered severe associated extra-abdominal injuries. Combined splenic and hepatic injury carried the highest mortality rate (88.9%). However, this high mortality does not appear to be due to the double intra-abdominal injury itself, but rather to the fact that such double injury occurred in patients with very extensive trauma and multiple injuries to other organ systems.

Summary

Blunt abdominal trauma is a grave surgical emergency requiring the utmost skill in its diagnosis and management.

The organs injured in order of frequency are the spleen, the liver, the gastrointestinal tract and the pancreas.

In patients with blunt abdominal trauma, the diagnosis of specific organ injury is often possible on a basis of a careful history and physical examination.

The leukocyte and differential counts are often more helpful in the diagnosis of solid organ injury with hemoperitoneum, than is the hemoglobin estimation.

Since these lesions are almost invariably fatal if not treated surgically, an aggressive surgical approach is mandatory. One should be prepared to accept the occasional negative laparotomy done for a contusion of the abdominal wall or retroperitoneal hematoma, in order to salvage those patients with lesions amenable to surgical correction.

Mortality figures, although interesting, mean little in the care of the individual patient and an aggressive program of resuscitation and surgical management will occasionally be rewarded by patient survival following wounds that appear incompatible with life. This fact must constantly be borne in mind in considering

whether or not to operate upon a patient with an apparently hopeless prognosis.

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RÉSUMÉ

L'auteur passe en revue une centaine de cas de lésions intra-abdominales majeures, survenues consécutivement, mais qui ne comprenaient pas de lésions des voies urinaires. Les organes blessés étaient, dans l'ordre de la fréquence: la rate, le foie, le tractus gastro-intestinal et le pancréas. Les accidents d'automobile ont été la cause de 62% de ces lésions mais, parmi les lésions spléniques, le traumatisme était parfois étonnamment faible. Une anamnèse précise et un examen somatique complet trouvent leur récompense dans l'obtention d'un diagnostic très précis des lésions spécifiques des organes. Neuf patients qui présentaient une lésion splénique ont été classés comme souffrant d'une rupture retardée. Le problème diagnostique d'une lésion intra-abdominale qui se présente chez un blessé à la tête, inconscient, est discuté et il comprend deux malades souffrant d'une péritonite biliaire post-traumatique consécutive à une lésion hépatique. L'auteur évalue la valeur diagnostique des analyses de laboratoire et des films radiographiques préliminaires. Il étudie en détail un plan pour les soins généraux à donner à un patient soupçonné d'avoir subi une blessure abdominale interne, la conduite de la laparotomie dans ces circonstances et le traitement d'une lésion organique spécifique. Trente-deux des 53 malades qui ont subi une splénectomie eurent des numérations en série de plaquettes et 25% d'entre eux avaient une thrombocytose supérieure à 1 million/c.mm. La mortalité globale a été de 17%, comportant de grosses variations parmi les groupes selon la nature spécifique de la lésion intra-abdominale et le nombre et l'ampleur des lésions connexes.

SCAR TISSUE CARCINOMA

More recently, attention has been focused on the epidermis-dermis relationships as it affects skin carcinogenesis. McGovern states that collagen degeneration in skin is due to the effects of ultraviolet light and plays an important role in carcinogenesis. This carcinogenic effect is mediated by its primary action on the dermis, thus altering the nutrition of the epidermis and somehow producing skin cancer . . . After reviewing the literature, Sevitt states that an abnormal dermis may play a role in cancer formation in scars . . .

Recently, Neuman summarized the four current possible etiologic mechanisms relating to the formation of cancer in scars. They are: (1) local environmental changes in both epidermis and dermis, (2) action of trauma as a co-carcinogen, (3) carcinogenic agents which may accompany the trauma, and (4) implantation of living epithelial elements into the dermis. Our experimental studies appear to support the theory that trauma acts as a co-carcinogen, with malignancy developing only on a rare occasion when some other unknown co-carcinogenic agent is also operative.—Arons, M. S. *et al.*: Scar tissue carcinoma: II. An experimental study with special reference to burn scar carcinoma, *Ann. Surg.*, **163:** 458, 1966.

CASE REPORTS

LES FAUX ANEVRYSMES POST-TRAUMATIQUES*

S. N. MARTINEZ, M.D., F.R.C.S.[C], C. BERTRAND, M.D., F.R.C.S.[C] et A. THIERRY, M.D., *Montréal, Qué.*

Les hématomes intra-crâniens associés à une rupture artérielle ont été rapportés par Vance,[1] Drake[2] et autres. Dans 10% des cas, les hématomes sont associés à une fissure extra- ou intra-durale d'une artère intra-crânienne. Il nous a paru intéressant de présenter dans ce rapport, trois cas d'hématomes intra-crâniens associés à une image angiographique d'un faux anévrysme post-traumatique.

Deux de ces cas ont une origine sur l'artère méningée moyenne, et s'associent à un hématome épidural. Le troisième cas est un hématome sous-dural résultant d'une fissure d'une artère corticale, avec un faux anévrysme à la région profonde de l'hématome.

Plusieurs cas de faux anévrysmes post-traumatiques des principaux vaisseaux de la base du crâne et de l'encéphale ont déjà été rapportés dans plusieurs publications.[3-12]

De très bonnes descriptions de rupture des carotides internes au niveau de l'os pétreux[9] et au niveau du sinus caverneux[5] ont été faites, mais il semble que la région supraclinoïdienne de la carotide interne soit la région la plus fréquemment touchée par cette variété de pathologie. Nous avons trouvé dans la littérature, cinq observations de faux anévrysmes post-traumatiques de l'artère méningée moyenne,[8, 13, 14] et trois cas se développant à partir d'une artère corticale.[8, 15] Chez tous ces patients, les faux anévrysmes se développent à l'intérieur d'une partie de l'hématome organisé autour d'une fissure artérielle.

Il est intéressant de noter que, lorsqu'on associe hématomes et faux anévrysmes, un délai important s'établit entre la présence de l'image angiographique anévrysmale et le moment du traumatisme.

*Service de neurochirurgie, Hôpital Notre-Dame, Montréal, Qué.

Hématome épidural et faux anévrysme de l'artère méningée moyenne

1er Cas.—A.L. est un patient de 35 ans, sans antécédents pathologiques, qui fit une chute avec traumatisme crânien léger, quatre jours avant hospitalisation; il a perdu connaissance pour quelque temps. Par la suite il développe une hémiparésie droite et une aphasie. Hospitalisé dans une autre institution pendant deux jours, il nous arrive au sixième jour après son traumatisme. A l'examen il présente une aphasie, une hémiparésie droite, une mydriase gauche, une hyperréflexie tendineuse et un Babinski bilatéral, une bradycardie à 54 pulsations à la minute, et une pression artérielle à 100/60. Il a un hématome orbitaire à gauche. La radiographie du crâne met en évidence une

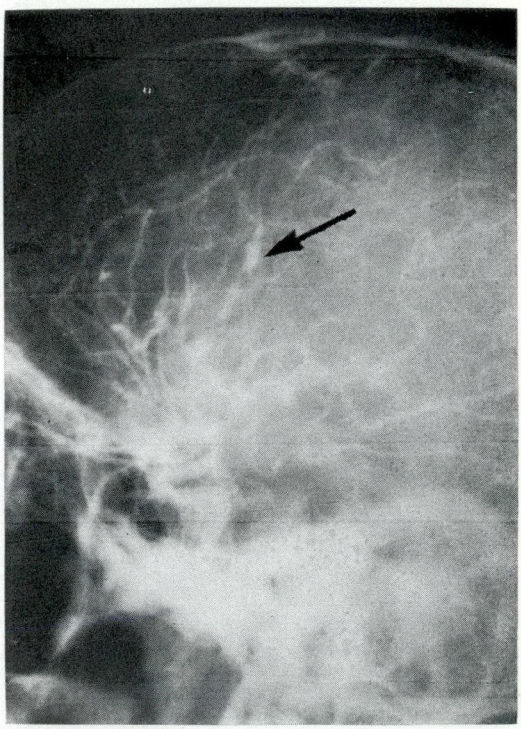

Fig. 1.—Artériographie carotidienne gauche montrant une image anévrysmale et une ectase artérielle à la région temporale dans le trajet de la méningée moyenne.

Fig. 2.—Artériographie antéro-postérieure montrant le déplacement de la cérébrale antérieure vers la droite, et la compression du groupe Sylvien vers le bas, dû à un hématome épidural.

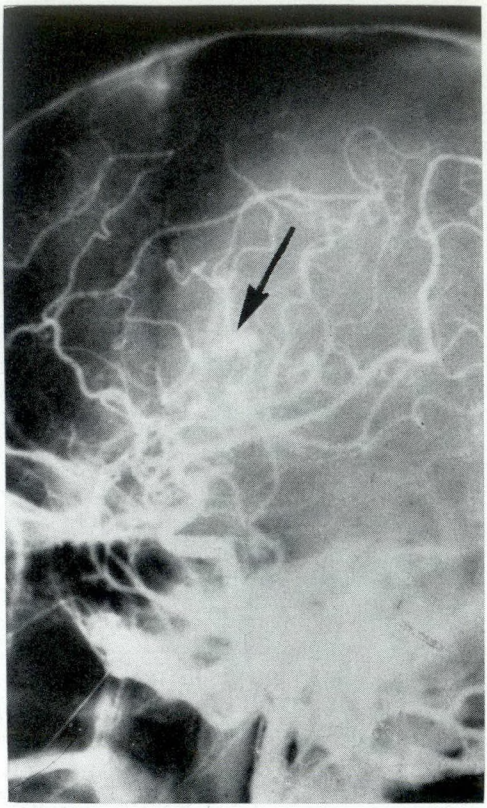

Fig. 3.—Artériographie de profile montrant une image anévrysmale au niveau de la région frontale temporale droite.

fracture linéale pariéto-temporale gauche. L'artériographie carotidienne gauche montre la présence d'un hématome pariétal gauche; les films en latéral montrent une ectasie anévrysmale dans le trajet d'une artère à la face profonde de l'hématome (Figs. 1 et 2).

A la crâniectomie on a pu constater la présence d'un hématome épidural pariéto-temporal gauche au fond duquel l'artère méningée moyenne présente une déchirure au niveau de l'emplacement de l'image angiographique anévrysmale. L'artère est coagulée et fermée à l'aide de clips. L'évolution post-opératoire a été satisfaisante, et le patient voit régresser rapidement son hémiplégie et son aphasie. Il reçoit son congé huit jours plus tard la récupération étant complète.

2e Cas.—C.J.P., un patient de 26 ans, fut impliqué dans un accident d'auto. Il présente des traumatismes multiples avec perte de conscience. Les reflexes aux quatre membres sont toutefois présents. Ses pupilles sont isocoriques; pendant deux jours, il demeure semi-comateux dans une autre institution. Il est transféré dans notre service et gardé sous observation durant trois jours; l'examen clinique reste inchangé. La radiographie du crâne ne montre pas de fracture. A la sixième journée, bradycardie à 54-60 et hypertension artérielle. Dans l'absence de signes localisateurs, on procède d'emblée à une artériographie carotidienne droite. Celle-ci révèle un hématome pariéto-frontal gauche au fond duquel une image d'anévrysme est visible. La crâniectomie nous a permis de constater la présence d'un hématome épidural fronto-pariétal gauche et à sa face interne, un caillot organisé sur une fissure de l'artère méningée moyenne (Figs. 3-5). Celle-ci a été fermée par un clip. Nous n'avons pas fait d'examen histologique chez ce patient, mais pour nous assurer qu'il ne s'agissait pas d'un anévrysme congénital de la corticalité, une artériographie de contrôle a été faite; elle n'a pas montré d'image anévrysmale. L'évolution post-opératoire a été simple, et le patient a reçu son congé sans signes déficitaires.

Hématome sous-dural et faux anévrysme post-traumatique d'une artère de la corticalité

3e Cas.—J.P., une patiente de 53 ans, fut impliquée dans un accident d'auto, sans trau-

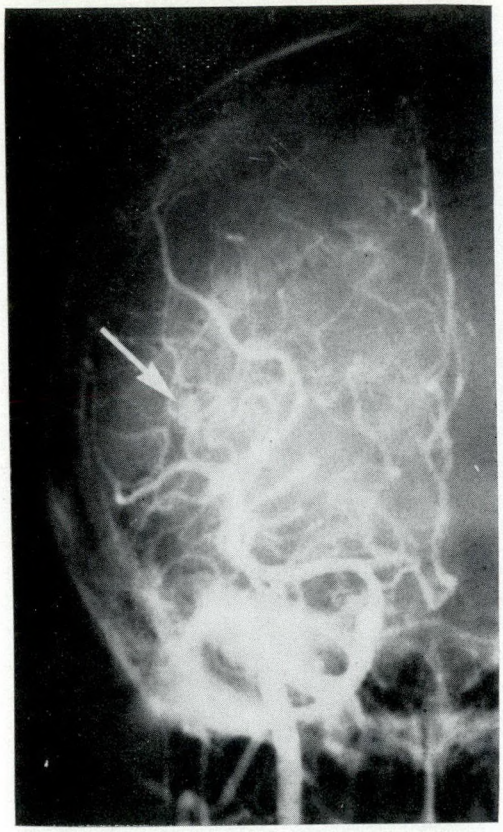

Fig. 4.—Vue antéro-postérieure démontrant une image anévrysmale avec déplacement vers le bas du groupe Sylvien.

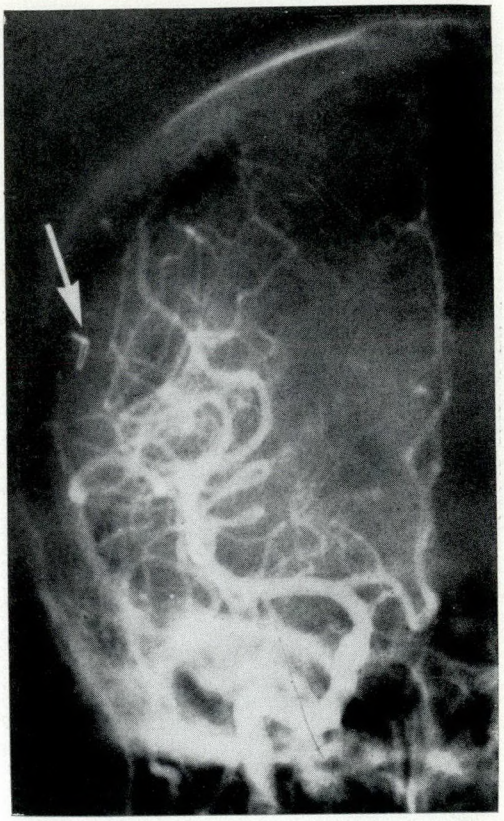

Fig 5.—Artériographie huit jours après l'intervention montrant le clip à l'endroit de l'image anévrysmale sur l'artère méningée moyenne.

matisme crânien important et sans perte de conscience immédiate. Quarante-cinq minutes après l'accident, elle se plaint de céphalée et d'étourdissement. A son entrée à l'hôpital elle devient somnolente et développe rapidement une hémiparésie droite avec anisocorie, la pupille droite étant plus grande que la gauche, avec dysphasie et Babinski bilatéral. Il s'agissait d'une patiente artérioscléroteuse avec une pression artérielle à 170/110; un électrocardiogramme suggère une ischémie antéro-septale. La radiographie du crâne est normale et l'électroencéphalogramme montre des ondes lentes des régions bitemporales sans autres particularités. Alors qu'elle présentait les signes cliniques déjà mentionnés, nous avons pensé faire une artériographie, mais deux heures après son entrée, la patiente s'éveille, son hémiparésie disparaît et ses pupilles deviennent symétriques. L'amélioration assez rapide nous a fait pensé à un phénomène de spasme artériel post-traumatique chez une artérioscléroteuse, et nous avons poursuivi l'investigation clinique tout en la gardant sous observation. Il a été démontré qu'il s'agissait d'une patiente diabétique, contrôlée médicalement. A la huitième journée d'hospitalisation alors que nous envisagions son congé, la patiente devient comateuse; l'examen neurologique montre à nouveau une hémiplégie droite, une dilatation pupillaire droite, et un Babinski bilatéral. Il était difficile de discerner s'il s'agissait ici d'une lésion donnant une hémiplégie ipsilatérale avec compression du tronc sur le bord libre de la tempe du cervelet. Une artériographie carotidienne a été faite, du côté de sa mydriase. Celle-ci a montré un important déplacement de la cérébrale antérieure et une image d'hématome sous-dural récent. A la face interne de cet hématome, nous constatons la présence d'une image anévrysmale sur la corticalité (Figs. 6 et 7). La condition générale de la patiente n'était pas très bonne, et comme il s'agissait d'une diabétique, nous avons décidé d'évacuer l'hématome sans toucher la formation anévrysmale. Trois trous de trépan ont été faits; l'hémotome sous-dural a été évacué et la patiente s'éveille sur la table d'opération. L'hémiplégie disparaît et l'évolution post-opératoire est satisfaisante. Huit jours plus tard, nous répétons l'artériographie,

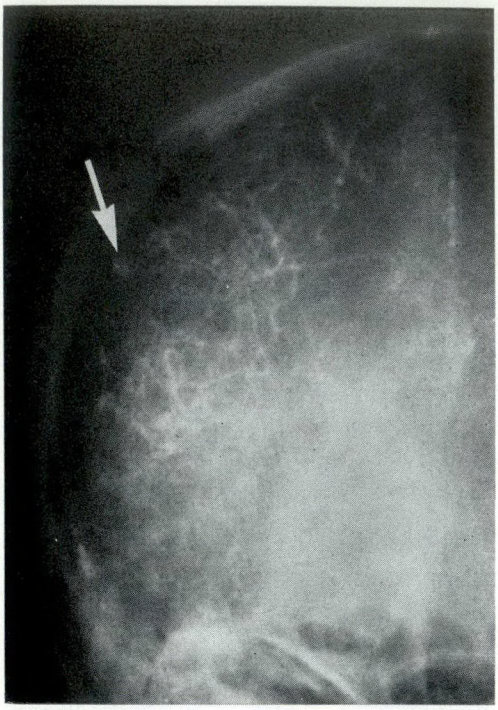

Fig. 6.—Radiographie de face, montrant une image anévrysmale à la région frontale postérieure et l'hématome sous-dural.

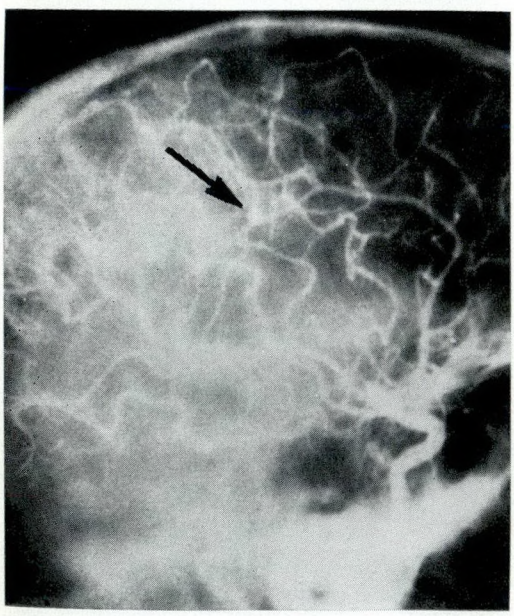

Fig. 7.—Vue de profile de l'image anévrysmale d'une artère de la corticalité.

ce qui confirme la présence de l'image anévrysmale. Sous anesthésie locale, un volet osseux à trois trous est rabattu, et nous constatons la présence d'un petit hématome organisé autour de l'artère corticale (Fig. 8). Celui-ci est extirpé en bloc, mais en le décollant, nous provoquons le saignement de la fissure artérielle. Deux clips de Mackenzie sont placés sur l'artère. L'évolution post-opératoire est sans incident. La patiente reçoit son congé une semaine plus tard, et par la suite, l'évolution se poursuit de façon normale.

Convaincu qu'il s'agissait d'un anévrysme post-traumatique, du même type que ceux décrits par Hirsch, David et Sachs,[8] nous avons disséqué le caillot. Une étude histopathologique nous a permis de constater qu'il s'agissait en réalité d'un faux anévrysme, dont la paroi était formée par des couches de fibrine richement infiltrée de cellules sanguines. Il n'y avait pas d'éléments de la paroi artérielle normale dans cette malformation anévrysmale.

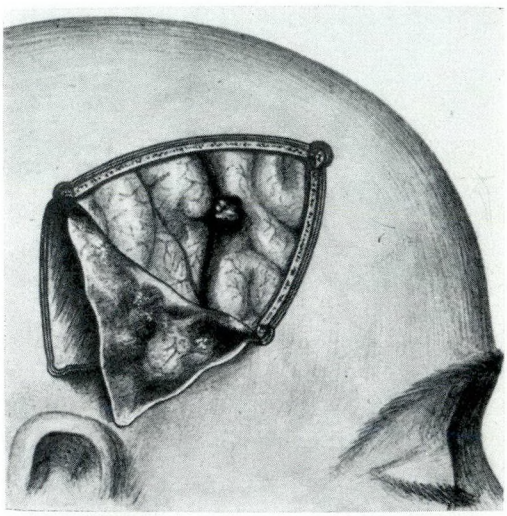

Fig. 8.—Figure schématique de l'image d'anévrysme lors de l'intervention.

Discussion

Ces trois cas d'hématomes épi ou sous-duraux associés à des fausses images de malformation artérielle gardent une similitude remarquable avec ceux décrits par Hirsch, David et Sachs[8] en 1962, par Pouyanne *et al.*[14] et par Ishii *et al.*[15] Cette malformation semble avoir comme origine, le même mécanisme que celui décrit par Drake[2] pour les hématomes d'origine artérielle (fissure d'une artère épidurale ou corticale). La paroi artérielle distendue brusquement à son point de contact dural intra- ou extra-crânienne, se fissure. Cette fissure entraîne un saignement lent mais progressif

et la formation de l'hématome épi- ou sous-dural.

La portion de l'hématome au pourtour de l'artère se laisse excaver par le remous sanguin qui le pénètre, mais la fibrine du caillot s'organise en mince couches et se laisse pénétrer par des macrophages et des hysthiocytes; ainsi se forme à la longue, une poche anévrysmale sans parois artérielles proprement dites.

Comme dans les cas décrits par Drake[2] dans les hématomes sous-duraux d'origine artérielle, un délai important existe entre le traumatisme et l'organisation morphologique de ce faux anévrysme. Il est intéressant de noter que la diminution de la vitesse à l'intérieur de l'image du faux anévrysme laisse celle-ci visible à la phase veineuse ou tardive de l'angiogramme (Fig. 9).

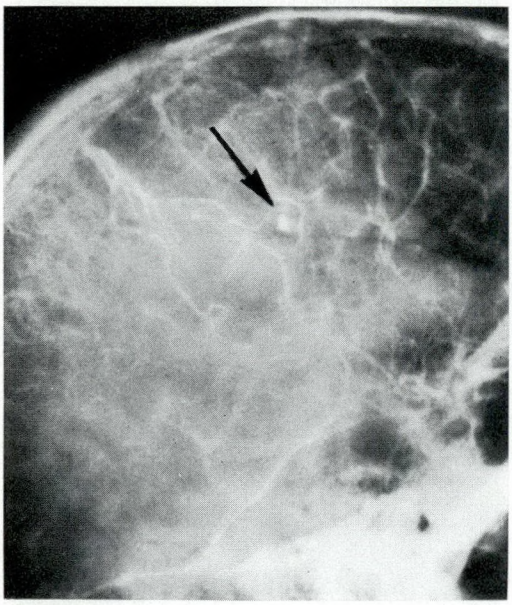

Fig. 9.—Phase veineuse de l'angiographie avec image anévrysmale encore visible.

Le traitement chirurgical doit être fait d'emblée dans les cas d'hématomes épiduraux, mais quand il s'agit d'un hématome sous-dural et d'un faux anévrysme chez un patient en très mauvaise condition, on peut se permettre de faire dans un premier temps, l'évacuation de l'hématome, et par la suite, dans de meilleures conditions de fermer l'artère en cause, et d'exciser le faux anévrysme. Cette technique a été appliquée dans notre troisième cas, avec un résultat des plus encourageants.

Résumé

Les anévrysmes post-traumatiques intracrâniens s'avèrent une rare entité nosologique en neurochirurgie. Par contre, il faut penser à sa présence lorsque l'angiographie nous montre une image anévrysmale à la face profonde d'une hématome épi- ou sous-dural. Sa forme est capricieuse; dans notre premier cas, elle apparaît comme une ectasie du vaisseau mais dans le deuxième et le troisième cas elle a définitivement l'aspect d'une poche anévrysmale.

Il nous semble important de reconnaître un faux anévrysme chez un traumatisé crânien, car un patient porteur d'une malformation anévrysmale qui subit un traumatisme crânien léger peut présenter un hématome ayant comme point de départ une rupture anévrysmale. Tout cela nous place dans une position médico-légale importante à reconnaître, car c'est l'étude histo-pathologique de la malformation qui tranche de façon définitive la relation des causes à effet, entre la rupture anévrysmale avec hématome subséquent et la rupture artérielle post-traumatique avec hématome organisé autour, donnant l'image angiographique du faux anévrysme post-traumatique.

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Summary

The three cases described in this paper are similar to those reported by Hirsch, David and Sachs in 1962, Pouyanne *et al.* and by Ishii *et al.* Hematoma formation in this particular case differed from the usual venous rupture after injury and brain contusion in that a fissure probably developed in the arteriosclerotic wall of a cortical artery perhaps because it had been overstretched. As leakage progressed, a clot formed as a thin-walled pouch into which blood was whirling; this may explain the angiographic appearance of an aneurysm. As in the cases reported by Drake, there may be some delay before the clot assumes the appearance of an aneurysm. As a matter of fact, local blood flow seems to be diminished because the appearance of the aneurysm is still present in the venous phase of the angiogram. Pouyanne and Hirsch reported an analogous aneurysmal formation arising from the meningeal artery after trauma; it had the histological appearance of a clot in which no normal artery wall was found.

Evacuation of the subdural hematoma is recommended as a first step, if the patient's condition does not permit a more extensive surgical procedure. Then, after recovery, the artery should be explored and clipped in a second stage. In two patients reported by Hirsch, extensive craniotomy was the first attempt at treatment; both patients died. The conservative approach—two-step surgery—in the present case may have been responsible for the excellent outcome.

Finally, it is most important to recognize the occurrence of false aneurysms produced by the head injuries because of the possible medicolegal implication.

OSTEOMA OF THE MASTOID

JOSEPH P. FLEMING, M.B., B.Ch., F.R.C.S.[C], F.A.C.S.,* *Vancouver, B.C.*

Approximately 39 cases of osteoma of the mastoid have been reported in the English medical literature. Simpson[1] in 1940 recognized that this tumour was rare and reviewed the then-existing literature (30 cases), and added two of his own. Single case reports have been published since 1940 by Cinelli,[2] Jervey,[3] Marrocco,[4] Neil,[5] Seltzer,[6] Chase[7] and Schwartz.[8]

An osteoma of the mastoid, which obstructed the external auditory canal and was associated with an osteoma and with hypertrophy of the right mandible, was recently encountered and is the subject of this communication.

Case Report

L.S., a white female, was first admitted to hospital in February 1962, at the age of nine years (Figs. 1 and 2).

Her chief complaints, a protruding right ear and preauricular "fullness", had first been noticed at the age of three years. She had associated complaints of occasional right earaches and discharge from the right external auditory meatus.

Examination revealed a firm swelling at the posteroinferior aspect of the right external auditory meatus, which was almost completely collapsed. There was asymmetry of the lower third of the face. The right hemimandible was relatively enlarged and was 1.5 cm. longer than the left hemimandible, measured between the mandibular angles and the midpoint of the symphysis. There was an associated crossbite (an occlusal derangement in which there is a disturbance of the normal width relationship between the upper and lower dental arches. Normally the buccal cusps of the maxillary posterior teeth and cusp of the canine tooth overlap the lower dentition, and are in a more buccalward and labialward position; with a crossbite this position is reversed. A unilateral crossbite may be produced as in the case presented when the mid-sagittal relationship of the

*Plastic Surgery Section, St. Paul's Hospital, Vancouver, B.C.

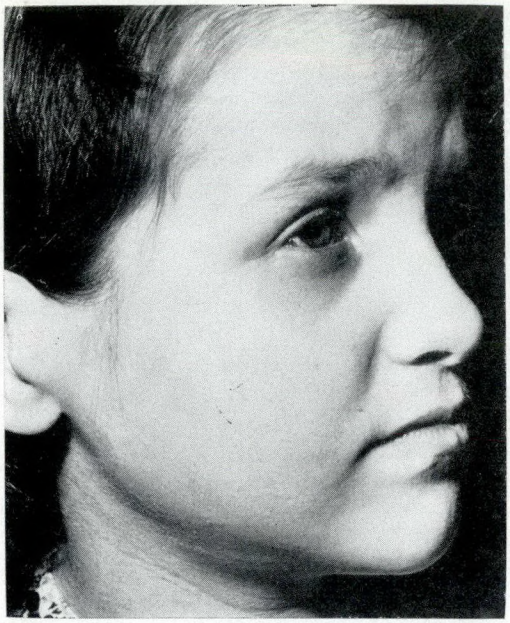

Fig. 1.—Preoperative photograph demonstrating deformity.

Fig. 2.—Preoperative photograph demonstrating mandibular deformity.

upper and lower dental arches is disturbed by an enlarged and deviated mandible.).

On February 26, 1962, through a right preauricular incision, a biopsy excision was done under general anesthesia. A mass, which was in continuity with the mastoid process, was found compressing the external auditory canal. A representative biopsy was taken and on subsequent microscopy the tissue was identified as a benign "osteoma". It was decided to delay definitive surgery until the patient was older.

In January 1965 the patient was readmitted and the results of laboratory investigation were as follows: urinalysis, normal; hemoglobin, 12.7 g.%; alkaline phosphatase, 16 Bodansky units; serum calcium, 9.8 mg.% and phosphorus, 4.1 g.%. A skeletal survey showed no evidence of bony pathology. Radiography of the temporomandibular joints showed relative restriction in the forward movement of the right condyle on the eminence; otherwise the joints were normal. The previously noted crossbite and the mandibular asymmetry were still present. The mandibular symphysis was protuberant and projecting to the left (Fig. 2). There was a fullness at the lower pole of the right auricle, the external auditory meatus was collapsed and hearing in the right ear was impaired.

On January 21, 1965, exploration was carried out through a preauricular incision extending into the postauricular region over the mastoid process and into the upper neck crease. The facial nerve was identified in the sulcus between the anteroinferior margin of the bony meatus and the anterior border of the mastoid process. A bony tumour was found involving the mastoid process and the bony external auditory canal. The tumour was excised in a plane 5 mm. lateral to the stem of the facial nerve. The temporomandibular joint was

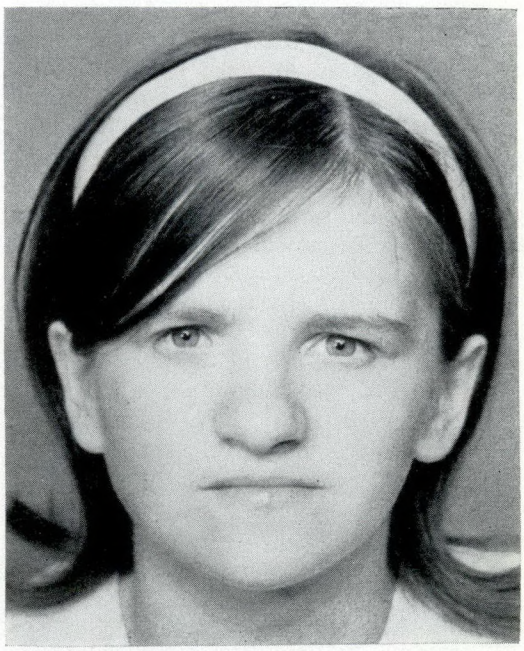

Fig. 3.—Postoperative photograph showing improvement in facial contours.

inspected including the zygoma; both were normal.

On subsequent microscopy the tumour was shown to be a benign compact-type osteoma (osteoma compactum).

On February 4, 1965, the protuberant mandibular symphysis was corrected. Under general anesthesia, through a buccal sulcus incision, the mandible was exposed subperiostealy and bilaterally, 1 cm. proximal to the mental foramina. An "osteoma" measuring 1.5 cm. in diameter was identified at the symphysis. There was a relative enlargement of the right symphysis in all dimensions. The tumour was excised with a margin of a hypertrophied mandible thereby recontouring the symphysis, and eliminating the asymmetry and protuberant appearance of the chin (Fig. 3).

Histology of the osteoma was similar to that of the mastoid.

Discussion

Simpson's paper[1] included individual abstracts of the 30 cases reported to 1940. Stuart,[9] whose case report is included in the above series, clearly defined the term "osteoma of the mastoid" as a benign, circumscribed, slow-growing tumour of bony mastoid tissues. Neil[5] emphasized the neoplastic nature of the growth in contrast to a reactive type of tissue response. Cinelli's pathological classification[2] included the term "osteoma compactum", which is the type most frequently encountered; it is hard, ivory-like and spherical, is attached to the cortex of the mastoid process but may penetrate into the mastoid cells. Histologically, the compact osteoma has lamellated bony tissue, which is transversed by a few vessels. "Osteoma cancellere" is a rare type and is composed of cancellous bone and fibrous cellular tissue. "Osteoma cartilagineum" is not uncommon and consists of bone cartilage. Marrocco[4] described a fourth type, which he called "osteoma mistum", which is a mixture of "osteoma cancellere" and "osteoma compactum".

According to most authors the osteoma originates from preosseous connective tissues. The tumour occurs most frequently in postpubertal women. It is slow growing and may produce external deformity and pressure symptoms, usually pain, which is referred to the auricle, the drum or the neck. The tumour may infiltrate the cortex well forward and cause meatal obstruction producing a conduction-type deafness as in the case presented. Thirty per cent of cases are associated with chronic suppuration, however this is regarded as secondary to the tumour. Radiographs may show a relatively radiolucent or rarified area of bone surrounded by what appears to be a dense zone. Metabolic disturbances should be excluded as suggested by Stuart,[9] whose patient had a family history of dyspituitarism. Stuart emphasized, as other authors do, that an osteoma of the mastoid is distinct from the common aural exostosis. He referred to Cheatle[10] who pointed out that aural exostosis originated invariably from the tympanic portion of the temporal bone.

Surgical removal of the osteoma is advocated by all writers on this subject. In the cases reviewed by Simpson,[1] the dura was exposed on four occasions and the lateral sinus as often; however, no complications were recorded in the reported cases. In six of these cases, the external auditory meatus was obstructed by the tumour, and this was a prominent feature in the present case.

Summary

A case of osteoma of the mastoid associated with an osteoma and unilateral hypertrophy of the mandible is presented. A brief review of the literature is included.

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Résumé

L'auteur décrit un ostéome de l'apophyse mastoïde chez une fillette de neuf ans; cette tumeur obstruait le conduit auditif externe et était accompagnée d'une hypertrophie du mandibule droit. Une biopsie effectuée en 1962 avait révélé que la tumeur était un ostéome bénin. On recula jusqu'au 19 janvier 1965 l'intervention radicale. La tumeur osseuse affectait l'apophyse mastoïde et la partie osseuse du conduit auditif externe; elle fut excisée dans un plan externe à la racine du nerf facial. En février 1965, par une incision du sillon buccal inférieur, la symphyse mandibulaire fut reconstituée par excision d'un ostéome qui occupait cette région.

On trouve dans la documentation anglaise environ 39 cas d'ostéome de l'apophyse mastoïde. Il importe de distinguer ces tumeurs des exostoses auriculaires courantes qui proviennent toujours de la portion tympanique du temporal. On les a groupées en "osteoma compactum", "osteoma cancellere", "osteoma cartilagineum" et "osteoma mistum". Elles proviennent de tissu conjonctif préosseux. Cette tumeur se rencontre fréquemment chez la femme durant la période post-pubertaire. Elle croît lentement et peut provoquer des douleurs par pression sur le pavillon, la membrane du tympan ou le cou. Une occlusion du méat, accompagnée de surdité du type conduction peut survenir, comme dans l'observation présente. Dans 30% des cas, on constate une suppuration secondaire. On conseille l'excision chirurgicale de la tumeur. Aucune complication de cette opération n'a été signalée.

LYMPHATIC CYSTS OF THE MESENTERY*

E. J. HINCHEY, M.D., C.M., M.Sc., F.R.C.S.[C],† *Montreal, Que.*

Cysts of the mesentery are rare intra-abdominal tumours. Benevieni, the Florentine anatomist, described the first mesenteric cyst in 1507. Since that time just over 600 cases have been reported in the literature. The largest single series, 13 cases, was described by Gross[1] in the 1953 edition of his textbook; most of the other reports included from one to three cases.[2-5] Steinreich[6] found one mesenteric cyst per 55,542 admissions to four Akron (Ohio) hospitals. Beahrs and Judd[7] at the Mayo Clinic found only seven chylous cysts among more than one million patients seen at that clinic. Burnett, Rosemond and Bucker[8] in 1950, reviewed 200 cases from the literature, 93 of which occurred in the small-bowel mesentery; 25% of the mesenteric cysts in this series occurred in children of 10 years or less. In the last 10 years, at The Montreal General Hospital, only three solitary cysts of the small-bowel mesentery have been recognized; these are the subject of this communication. During the 10-year period there were 184,377 admissions to The Montreal General Hospital, giving an incidence of one cyst per 61,459 admissions.

Case Reports

Case 1.—A 30-year-old woman was referred to hospital by her family physician because of anorexia and a mass in the left upper quadrant. She had complained of an intermittent burning pain and tenderness in the left upper quadrant and low midthoracic back pain for seven months before admission. The pain was not related to the ingestion of food or to bowel function, was more severe late in the day and in the early evening and was aggravated by bending forward. She had had anorexia for some months and had lost 20 lb. in weight. There had been no change in bowel habit. She got some relief by lying flat on her stomach. There was no history of nausea or vomiting. She admitted to a feeling of fullness in the epigastrium after the ingestion of small amounts of food.

A tender 10-cm. firm mobile mass could be palpated in the left upper quadrant, close to the midline; it descended to the level of the umbilicus on full inspiration. Physical examination was otherwise negative. The patient was afebrile on admission, had a pulse of 80/min. and a blood pressure of 100/60 mm.Hg. The hemoglobin was 12 g.% and the hematocrit 38%. The white cell count was 6900/c.mm. with a normal differential. Platelets were 209,000/c.mm. and the prothrombin time was normal. An upper gastrointestinal (GI) series was done, which showed a deformity of the greater curvature of the stomach due to extrinsic pressure (Fig. 1). On barium enema, the transverse colon appeared to be indented by an extrinsic mass lying above the colon, just to the left of the midline. Intravenous pyelograms with standing films revealed normal nonmobile kidneys. Radiographs of the cervical and upper thoracic spines were normal.

At laparotomy, a firm, white, round mass, 6

*From the Department of Surgery, The Montreal General Hospital and McGill University, Montreal, Que.

†Assistant Professor of Surgery.

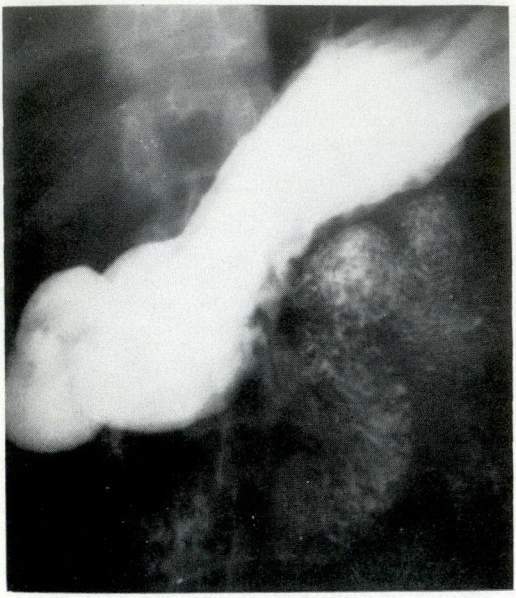

Fig. 1.—Gastrointestinal series showing indentation of greater curvature of stomach by extrinsic mass (Case 1).

cm. in diameter, was found in the base of the mesentery of the first loop of jejunum, overlying the superior mesenteric artery and vein as they emerged from behind the pancreas over the third part of the duodenum (Fig. 2). The middle colic artery and vein were adherent to the upper aspect of the mass. There was edema and fibrous tissue reaction in the mesentery around the tumour. Laparotomy was otherwise normal. The cyst was aspirated and caseous sebaceous-like material obtained (Fig. 3). The cyst was opened, evacuated and enucleated from the mesentery. The patient made an uneventful recovery. Bacterial cultures and guinea-pig innoculation of the contents of the cyst were both negative.

The wall of the cyst was 2 mm. thick and was composed of fibrous connective tissue. There was no lining. The pathological diagnosis was that of simple cyst of the mesentery.

Case 2.—A 60-year-old white man was admitted to hospital because of increasing constipation and abdominal pain. The abdominal pain was dull and continuous in both lower quadrants. He also had had low back pain for five weeks before admission. The patient had a poor appetite and had lost 12 lb. since his abdominal complaints began. Urinary frequency and urgency during the day had been present for the same five weeks. Micturition aggravated the lower abdominal pain.

The patient was alert, had a blood pressure of 130/80 mm.Hg, a pulse of 84/min. and normal temperature. A freely mobile round mass was felt in the abdomen midway between

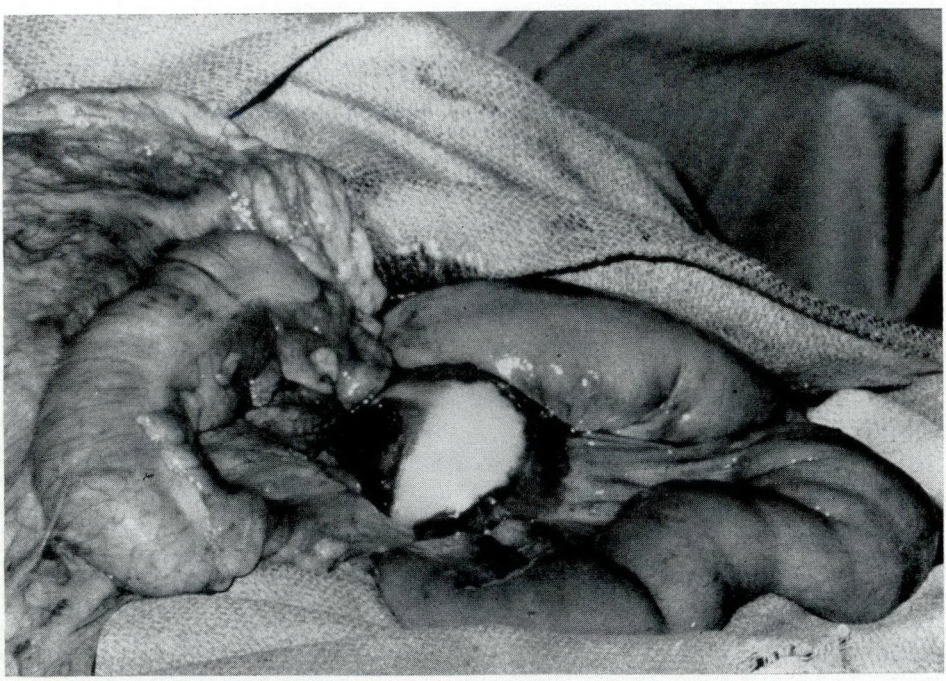

Fig. 2.—Transverse colon and omentum displaced cephalad to show cyst in mesentery of first jejunal loop.

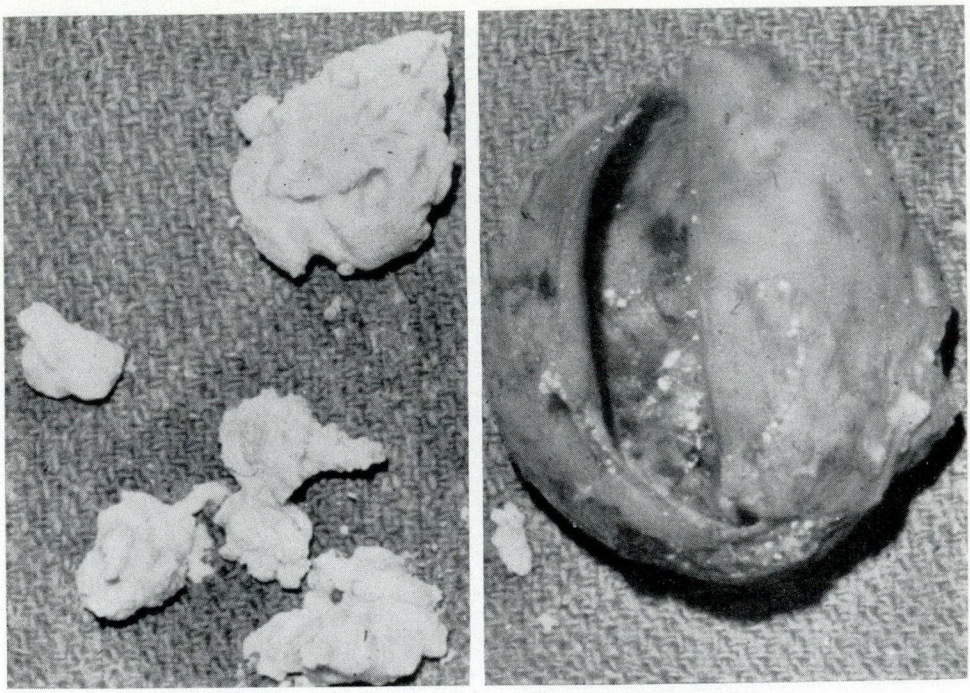

Fig. 3.—Opened specimen with caseous contents.

the umbilicus and pubis, which was tender on deep palpation. The hemoglobin was 13.5 g.%; hematocrit 42%; sedimentation rate 36 mm.; white cell count 6300/c.mm. with a normal differential. Three stool specimens were positive for occult blood. A flat plate of the abdo-

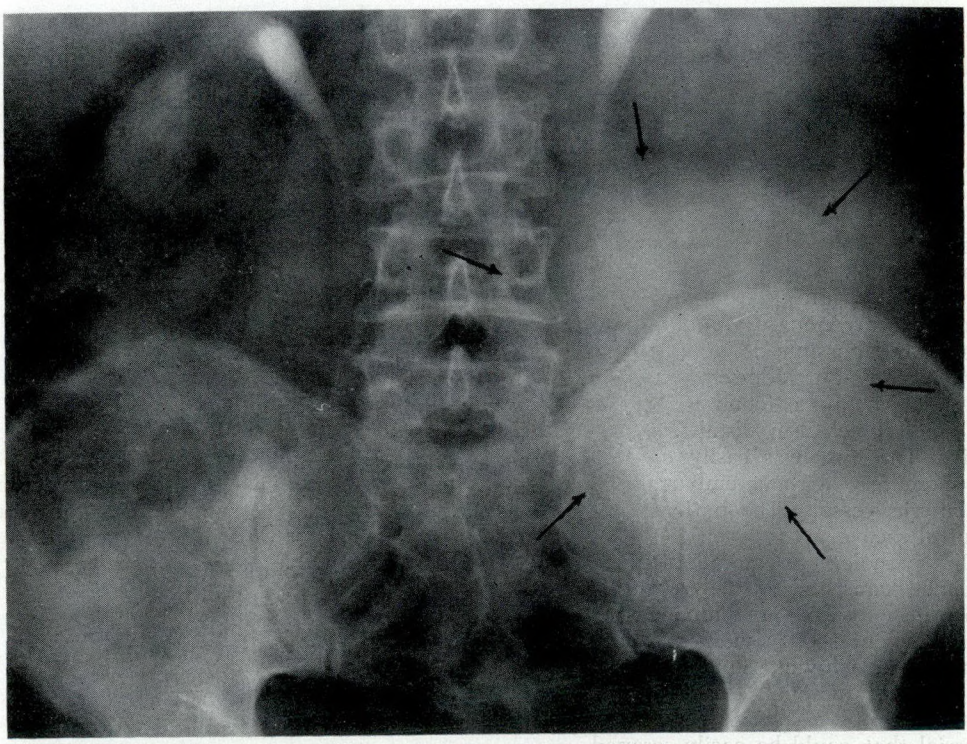

Fig. 4.—Soft-tissue mass in lower abdomen to the left of the midline (Case 2).

men showed a circular soft-tissue mass lying just to the left of the midline within the pelvis, which measured 11½ cm. in diameter (Fig. 4). No calcification was present. The barium enema was normal and there was no obvious extrinsic pressure on the colon by the intra-abdominal mass. Intravenous pyelograms revealed no abnormality of the kidneys or their drainage systems.

At laparotomy, a 10-cm. cyst was found in the mesentery of the jejunum, 2½ ft. from the ligament of Treitz. The external surface of the mass was reddish, mottled with white. Aspiration of the mass produced sebaceous-like material. Two feet of jejunum and the mesentery containing the cyst were resected. The patient made an uneventful recovery.

On pathological examination submucosal ectopic ductular tissue in the antemesenteric border of the jejunum (columnar epithelial ducts enveloped in smooth muscle) and an intramesenteric pseudocyst with intense surrounding inflammatory reaction in the fat, and fibrous occlusion of adjacent blood vessels were the principal findings.

Case 3.—A 28-year-old Chinese woman noted the onset of an epigastric mass with mild discomfort three months after the delivery of a normal child. Frequency of urination without dysuria had been present for the same period of time. The functional enquiry was otherwise negative.

Her blood pressure was 100/60 mm.Hg, her pulse 70/min. and her temperature was normal. The abdomen contained an easily palpable epigastric mass, which was tender and mobile. The hemoglobin was 13 g.%; hematocrit 36%; white cell count 5700/c.mm. with 78% neutrophils. An upper GI series revealed an indentation of the greater curvature of the stomach by an extrinsic mass in the left upper quadrant. Barium enema examination was normal. Intravenous pyelograms were normal except for a possible deformity of the superior calyx of the right kidney. At operation, a 10-cm. cystic mass was found in the mesentery of the small bowel, 2 in. distal to the ligament of Treitz. The cyst was easily enucleated from the mesentery without embarrassment to the mesenteric vessels. The patient made an uneventful recovery.

On pathological examination, the gross specimen consisted of an irregular structure measuring 7 cm. in diameter, which was fluctuant. Cut section yielded a large amount of milky greyish-white fluid with some colourless grumous material. The lining was smooth and covered by a yellowish cottage-cheese-like material that could be easily scraped off. The wall in the thickest portion measured 6 mm. and in the thinnest portion 1 mm. and appeared to be fibrous for the most part.

The sections of the cyst revealed an outer rim of fibrolymphoid and an inner rim of fibrocollagenous tissue. The inner surface was entirely ulcerated and lined by granulation tissue containing foamy phagocytes but with no preserved cellular membrane. The chylous content and the presence of lymphoid tissue strongly suggested that this was a lymphatic cyst. It was concluded that this was a chylous cyst of the mesentery, ulcerated but presumably lymphatic in nature.

Discussion

Simple cysts of the mesentery are rare. Most authors accept Gross's concept that they arise from sequestrated lymphatic tissue, which does not communicate with the remainder of the lymphatic system and which proliferates and accumulates fluid.[1, 9] The contents may be serous or chylous, the former being by far the more common. Dehydration of the chyle leaves a caseous, sebaceous material, as in two of our cases. The cyst in Case 3 probably represents a transition phase between a typical chylous lymphatic cyst and a pseudocyst containing grumous sebaceous material. It is considered unlikely that these cysts arise from traumatic or inflammatory obstruction of the mesenteric lymphatics with secondary cyst formation. Solitary cysts are by far the most common but multiple cysts do occur. Callaghan and Farley[10] collected seven cases of multiple mesenteric cysts and stressed the high incidence of associated abnormalities of lymphatic formation and intestinal rotation; these are uncommonly associated with single cysts. Amos[9] pointed out the similarity of multiple lymphatic mesenteric cysts to lymphangiectasia elsewhere in the body and suggested that mechanical obstruction might lead to rapid cystic dilatation of congenitally dilated lymphatic spaces; secondary proliferative changes in the pericystic connective tissues may contribute to their size.

Many authors emphasize the importance of distinguishing between simple cysts of the mesentery and duplications of the gut.[6, 11-14] The cystic mass in duplication of the gut contains all three layers of normal bowel in its wall and may or may not com-

municate with the intestinal tract. Beardmore and Wiglesworth[15] and Veeneklaas[16] showed that, in the presomite embryo, an adhesion may form between the ectodermal and entodermal germ layers, which may split or deviate the notochord. This results in the formation of vertebral anomalies and alimentary system traction duplications. The latter may occur in the posterior mediastinum between the layers of the mesentery or in both locations. These same authors also point out that radiographs of the chest and cervical thoracic spine should be made in patients with possible duplication of the gut. Conversely, patients with the Klippel-Feil syndrome—anterior spina bifida, posterior spina bifida, congenital scoliosis, hemivertebra, under-segmentation, under-development or partial fusion of the cervical or dorsal vertebra—should be examined for possible duplication of the gut. A review of the various theories of embryogenesis of duplication was presented by Inouye *et al.*[17]

In the series by Burnett, Rosemond and Bucker,[8] the most common complaint of patients with mesenteric cysts was abdominal pain. Anorexia, nausea, vomiting and weight loss are not uncommon clinical features. Frank intestinal obstruction may occur if the small bowel is tightly stretched over the surface of a tense mesenteric cyst. Two of our patients had urinary frequency presumably due to extrinsic pressure on the urinary bladder. A palpable mass is present in 60% of cases but its absence does not rule out the diagnosis. Handelsman and Ravitch[11] point out that thin-walled cysts may not be tense enough to be palpated. Other factors, such as the size of the mass, the position in the mesentery and the thickness of the abdominal wall may make palpation difficult. Moore[18] described a nine-year-old girl who had been treated for ascites by her family physician for 18 months before a large mesenteric cyst was recognized at laparotomy. Paracentesis had yielded 1300 c.c. of fluid, which had reaccumulated by the time of operation. As a form of treatment, aspiration of these cysts is mentioned only to be condemned.

The diagnosis of mesenteric cyst should be suspected when a patient presents with nondescript abdominal pain, the presence or absence of which may be related to posture. Back pain due to traction on the mesentery is not an uncommon accompaniment. The performance of a GI series, barium enema and intravenous pyelogram rule out intrinsic abnormalities of the stomach, colon and kidneys. The free mobility of the cyst and its marked respiratory excursion further suggest the diagnosis. Radiographs of the chest and cervical thoracic spine should always be made to rule out mediastinal masses or vertebral anomalies, which, if present, suggest duplication of the gut rather than a mesenteric cyst. The cystic nature of the lesion may not be appreciated at operation if the wall is thick and the contents caseous. In two of our cases the cysts resembled solid tumours of the mesentery and the surrounding inflammatory reaction caused a puckering of the mesentery not unlike that seen in malignant infiltration. It is important to establish the benign nature of these lesions and avoid unnecessarily radical surgery; to this end we recommend aspiration with a large-bore needle and syringe, a most useful procedure. The operation of choice is enucleation, if this is possible without impairment of the blood supply to the adjacent bowel. If enucleation is impossible, resection of the wedge of mesentery containing the cyst and involved segment of small bowel should be carried out with end-to-end anastomosis. A 16% mortality was associated with resection in the series reported by Burnett in 1950; it is considerably less in more recent series.

Summary

Mesenteric cysts are uncommon, but probably not as rare as has previously been thought. Three cases were seen at The Montreal General Hospital from 1955 to 1965. Abdominal and low back pain, a palpable mass and weight loss were features in these patients. A careful history, appropriate radiographs and the awareness of these lesions should lead to a correct preoperative diagnosis. Aspiration may be necessary at the time of surgery to confirm the benign nature of the mass. Enucleation is the surgical treatment of choice, followed by resection if there is any impairment to the blood supply of the small bowel.

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RÉSUMÉ

Les kystes du mésentère sont de rares tumeurs intra-abdominales. La majorité des kystes simples prennent naissance à partir de tissu lymphatique et contiennent une substance chyleuse, séreuse et, parfois caséeuse épaisse. Les kystes multiples accompagnent souvent d'autres anomalies du système lymphatique et des vices de rotation intestinale. Des kystes mésentériques simples doivent être distingués des duplications intestinales auxquelles s'associent souvent des malformations vertébrales. L'auteur en rapporte trois cas qui ont été observés durant la dernière décennie à l'Hôpital général de Montréal.

Les signes cliniques les plus courants comprennent une douleur abdominale, de l'anorexie, de l'amaigrissement et une masse abdominale palpable et mobile. Des radiographies appropriées permettent d'éliminer les anomalies intrinsèques de l'estomac, du côlon et des voies urinaires. Si la nature de la lésion n'est pas évidente au moment de la laparotomie, il est souvent nécessaire de procéder à l'aspiration du kyste. L'énucléation est le traitement opératoire de choix, à condition qu'elle puisse être entreprise sans embarrasser la circulation intestinale. Il est parfois indispensable de reséquer le mésentère affecté et la portion adjacente de l'intestin. Bien que ces lésions soient rares, il importe de reconnaître la nature bénigne de ces lésions, de façon à éviter dans toute la mesure du possible une intervention radicale inutile.

AN ATLAS OF ORTHOPEDIC SURGERY. Lewis Cozen. 732 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1966. $16.50.

Many operative procedures in common use in the practice of orthopedic surgery are described throughout the 732 pages of this book. The contents are divided into 10 chapters dealing with the surgery of the hand, the elbow, the shoulder, the foot, the leg, the hip, the gluteal region, the spine, amputations and incisions. Each procedure is dealt with in the form of classified notes on the left-hand page and clear, concise diagrams on the right-hand page: the notes give the indication for the procedure, the technique of the operation and a short discussion. Though most of the operative procedures in common usage are dealt with in this clear concise manner, a volume of this size cannot include all procedures in use at the present time. This does not detract from the value of such a book, which should be of considerable help to residents in orthopedic surgery. The presentation in note form should help residents to be concise and to learn the consecutive steps of any operation in a logical manner. The simplicity and clarity of the diagrams should be of help to them in learning quickly what is involved in a particular operative procedure. In many instances the postgraduate student will likely need to refer to a textbook that considers each operation in greater detail after he has mastered the essentials set out in this book. Similarly, he will need to go to a larger textbook for discussion of some of the newer and more complicated procedures now in use; indeed, a number of these will be found in the journals rather than in any textbooks. This, however, does not in any way detract from the value of this textbook, which gives a clear and concise overall picture of the field of operative orthopedic surgery. The requirements of the resident starting to work in this field could scarcely be met in a better way. This reviewer wholeheartedly recommends the book for this purpose.

METASTASIZING BASAL CELL CARCINOMA: A CASE REPORT

ROBERT JACKSON, M.D., F.R.C.P.[C],* *Ottawa, Ont.*

Basal cell carcinoma (rodent ulcer) is a common new growth of the epidermis or adnexal structures. The vast majority of these tumours are only locally malignant.[1] The purpose of this communication is to report one patient with basal cell carcinoma who developed metastatic disease in the ribs, lymph nodes and vertebrae.

Dahlgren and Martensson[2] set down the following criteria for the diagnosis of metastasizing basal cell carcinoma: (1) The primary tumour must be localized to the skin. (2) It must be possible to show metastases in lymph glands or viscera. (3) The histopathological picture in both the primary tumour and the metastasis must show the

TABLE I.—Summary of 47 Cases of Metastasizing Basal Cell Carcinoma Reported to Date (Modified after Dahlgren and Martensson[2])

Author	*Year*	*Site of primary tumour*	*Localization of metastasis*
Beadles	1894	face	lymph node
Fordyce	1902	nose	lymph node
Korbl (Case 61)	1912	temple	lymph node
Hazen	1917	hand	lymph node
Hazen	1917	nose	lymph node
Hazen	1917	right axilla	lymph node
Hazen	1917	forehead	lymph node
Finnerud	1924	face	lymph node
Finnerud	1924	face	lymph node
Mulzer	1930	temple	lymph node
Mulzer	1930	temple	lymph node
Spies	1930	cheek	bones, lungs, spleen, liver
Louste *et al*	1931	forehead	zygoma
Goodman	1931	cheek	lymph node
Cade	1940	not stated	lymph node
De Navasquez	1941	forehead	lungs, bones
Singer	1945	face	bones
Streitmann	1946	face	lymph nodes
Amersbach	1947	scalp	lymph node
Foot	1947	face	lymph nodes
Small	1949	ear	lung, liver, spleen, kidney, pleura, peritoneum, lymph node
Small	1949	ear	lung, liver, spleen, kidney
Eckhoff	1951	scalp	lymph node
Lattes and Kessler	1951	wrist	lymph nodes
Lattes and Kessler	1951	scalp	lymph nodes
Michel	1955	neck	lung
Bogoslavskii and Skomorovskaia	1957	not known	skeleton
Huntington and Levan	1957	face	lung
Richter	1957	scrotum	lung
Rank	1958	forehead	lymph node, parotid gland
Pickren and Katz	1958	face	lung (aspiration)
Pickren and Katz	1958	face	lung (aspiration)
Cotran	1961	scalp	lymph node
Cotran	1961	face	lymph node, lung
Cotran	1961	scalp	lymph node
Cotran	1961	eyelid	left parotid region
Cotran	1961	face	lymph nodes
Cotran	1961	nose	lymph nodes
Cotran	1961	scapular region	lymph node, lung
Cotran	1961	eyelid	lymph node
Cotran	1961	scalp	lymph node
Raitschev *et al*	1961	scalp	ear
Binkley and Rauschkolb	1962	scapular area	lymph nodes
Lewin	1963	forehead	lymph nodes
Dahlgren and Martensson	1963	cheek	liver
Crawford and Joslin	1964	scalp	lung, spleen, liver, pancreas, dura, vertebrae
Jackson (present case)	1966	neck	lymph nodes, ribs, vertebrae

*Consultant dermatologist, Ontario Cancer Treatment and Research Foundation, Civic Hospital Division, Ottawa Clinic, Dr. G. T. Stoddart, Director, Ottawa, Ont.

typical form of basal cell carcinoma. There must be no signs of epidermoid differentiation.

The 47 cases reported to date in the medical literature[3-5] are summarized in Table I.

Case Report

J.E.W., a 70-year-old man, had a growth in the skin on the right side of his neck, which started when he was aged 61. At age 66 a 5 x 3 cm. ulceration was present over the midpoint of the right sternomastoid muscle. There was a great deal of induration and puckering of the skin, and fixation to the underlying structures. There was tethering of the neck on the affected side, giving the appearance of an acquired torticollis. Clinical diagnosis was rodent ulcer (basal cell carcinoma). No lymph node enlargement was noted. A biopsy (Fig. 1) confirmed the clinical diagnosis. Jaw cysts, bifid ribs, palmer and plantar punctate depressions, as found in the hereditary basal cell nevi syndrome,[1] were not present.

The initial treatment of the rodent ulcer on the right side of the neck was a radical block dissection. Material submitted for biopsy showed an extensive basal cell carcinoma, with much associated fibrosis, extending into the hypoderm. Basal cell carcinoma was also present in a submitted regional cervical lymph node (Fig. 2). It was not possible to determine whether the tumour in the lymph node arrived there by direct extension or represented a true

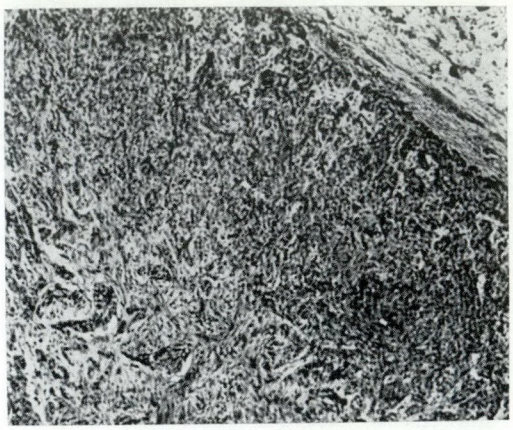

Fig. 2.—Basal cell carcinoma in lymph node from right side of neck.

Fig. 1a

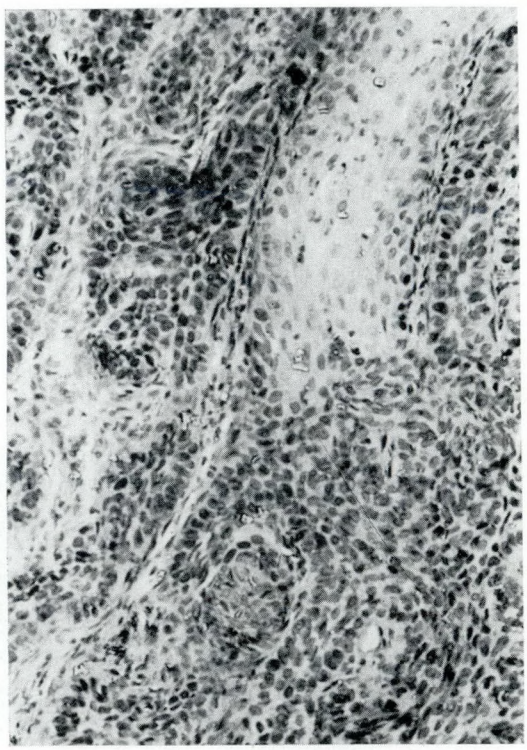

Fig. 1b

Fig. 1.—(a) Initial biopsy from skin right side of neck, showing basal cell carcinoma contiguous with the overlying epidermis. (All sections were stained with hematoxylin, phloxine and saffron.) (b) Detail of Fig. 1a.

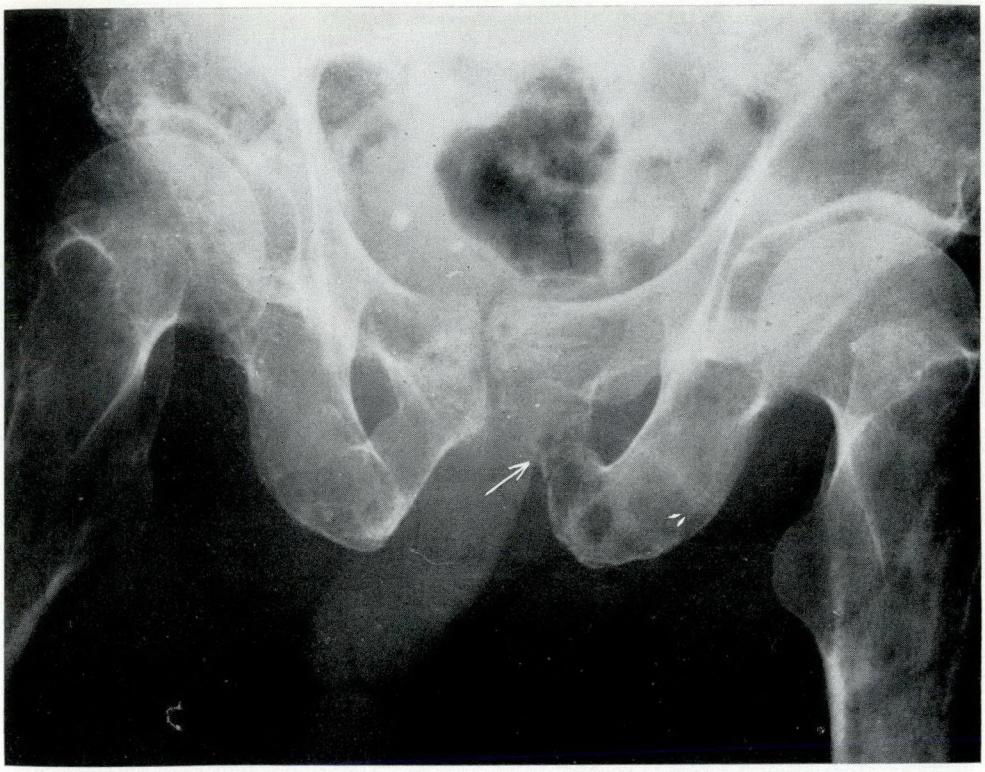

Fig. 3.—Radiograph of pelvis and femurs. Note destructive lesion in left pubic arch.

metastatic lesion. Eight months later the tumour was still present and another extensive surgical removal was attempted. This was followed by Co^{60} beam therapy to a total dose of 6100 r. At no time was the local lesion ever considered to be free of tumour.

At the age of 69 he was admitted to hospital because of back pain. Radiographs of the skull and ribs showed radiolucent areas. There was moderate osteoporosis of the thoracic and lumbar vertebrae, and marked compression of the eighth and tenth thoracic, and the first and fifth lumbar vertebral bodies. These findings were considered to be consistent with multiple myeloma or metastatic carcinoma. The final clinical diagnosis was osteoporosis and metastatic deposits in the vertebral column.

At age 70 he was admitted again to hospital because of low back pain and weakness. He was cachectic. Radiographs showed generalized osteoporosis and multiple osteolytic lesions of the ribs, vertebrae, pelvis (Fig. 3) and femurs. Examination of the skin of the right side of the neck showed a 3 x 3 cm. sharply demarcated ulcer with much sclerosis, atrophy and telangiectasia. One morning he suddenly became very ill and died a few hours later.

Post-mortem Examination

A complete autopsy was performed. Only the relevant findings are reported.

The cause of death was an extensive area of recent hemorrhage in the right cerebral hemisphere and in the pons. He also had

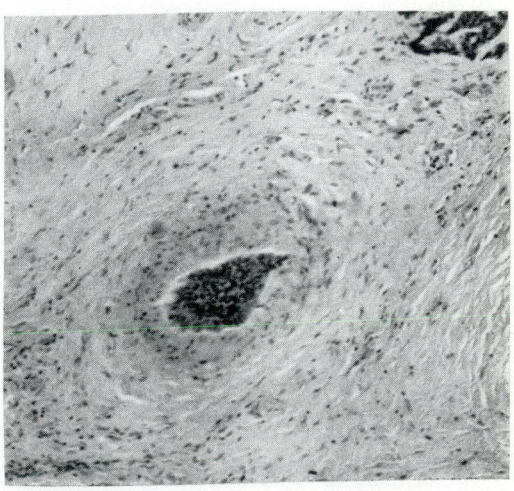

Fig. 4.—Basal cell carcinoma in blood vessel in lower dermis. Dense sclerotic fibrous tissue is also present.

marked atherosclerosis of the aorta and coronary arteries.

In the neck, dense sclerotic fibrotic tissue was seen, which contained basal cell carcinoma. Tumour was obviously present in many of the dermal blood vessels (Fig. 4). The vertebrae were massively invaded and in many areas

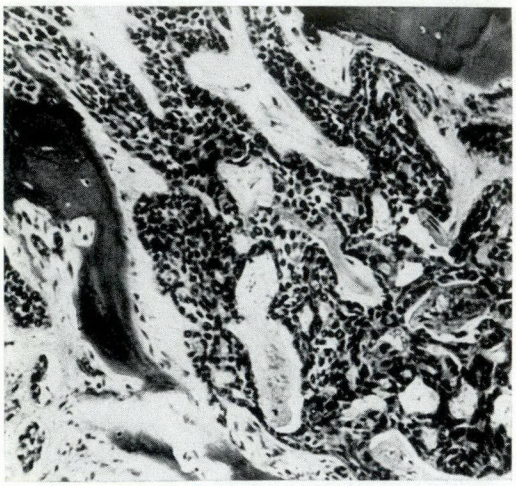

Fig. 5.—Verterbrae showing large amounts of basal cell carcinoma.

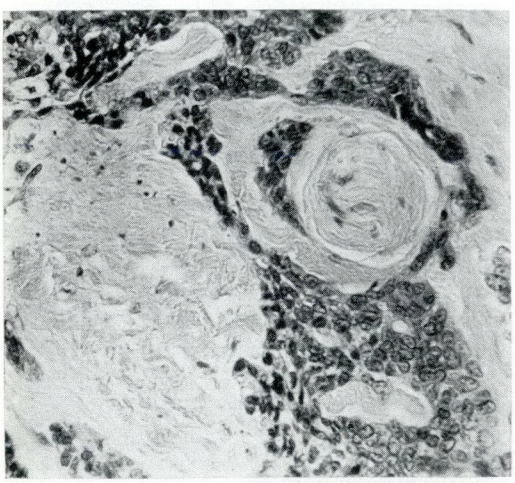

Fig. 6.—Basal cell carcinoma in perineural lymphatics about small nerve in perivertebral tissue.

replaced by basal cell carcinoma (Fig. 5). The lymphatics about many of the nerve trunks in the perivertebral tissue contained the same tumour tissue (Fig. 6). There was neither gross nor microscopic evidence of tumour in the prostate, thyroid, lung or adrenals.

Comment

This case fulfils the criteria previously outlined by Dahlgren and Martensson.[2]

The reason for the "aggressive" behaviour of a very small percentage of basal cell carcinomas is not known. Histologically they seem to have the same appearance as other "non-aggressive" basal cell carcinomas. Clinically they are frequently large lesions although this is probably an expression of either "virulence" or a lack of "host resistance".

Summary

The case protocol and autopsy findings are reported of a patient who showed metastatic lesions arising from a basal cell carcinoma in the skin of the right side of the neck. Metastatic disease was present in the ribs, lymph nodes and vertebrae.

The author wishes to thank Dr. John Jackson, Head of the Department of Pathology, National Defense Medical Centre, Ottawa, Ont. for his co-operation. Photomicrographs were taken by the Department of Photography, Ottawa Civic Hospital. Dr. K. Fani, formerly of the National Defense Medical Centre, performed the autopsy.

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Résumé

Nombreux sont les médecins qui croient que les cancers à cellules basophiles ne donnent jamais lieu à des métastases. Tel n'est pas pourtant le cas. On a signalé environ 50 cas où des métastases se sont produites et il y a peut-être un nombre égal de cas qui n'ont pas été rapportés. L'auteur décrit un patient qui était porteur d'un cancer du cou et de tumeurs métastatiques des côtes, des ganglions lymphatiques et des vertèbres.

EXPERIMENTAL SURGERY

ACUTE HEMORRHAGIC NECROSIS OF THE PANCREAS OF THE DOG AND ITS SUPPRESSION WITH TRASYLOL*†

G. B. OLSEN, M.D.‡ and
H. T. G. WILLIAMS, M.B., Ch.B., F.R.C.S.[Eng. & C], F.A.C.S., *Edmonton, Alta.*

FOR MANY years there has been much controversy over the role of trypsin in the development of hemorrhagic necrosis of the pancreas.[1] There is no other situation in the body where such a catastrophic pathologic picture is seen and it would seem inappropriate to dismiss lightly the significance of the presence of powerful pancreatic proteolytic enzyme systems as etiologic factors. While there is much evidence for and against trypsin as an etiological factor, to the present time it has never been conclusively demonstrated to be of prime importance in this respect.

Trasylol, a polypeptide with proteolytic enzyme-inhibiting properties, has recently been used as an adjunct in the treatment of acute pancreatitis. In the laboratory this drug has been shown to increase the survival rate of dogs with experimentally induced pancreatitis.[2] The mechanism of action is believed to be systemic and involves the inhibition or destruction of toxic products released by the disease process. If the necrosis seen in the pancreas during acute hemorrhagic pancreatitis is due to trypsin release, it should be possible to suppress the development of necrosis by delivering large quantities of Trasylol to the pancreas during the development of pancreatitis. To test this hypothesis a technique for inducing pancreatitis was required that would allow the process to develop gradually over a short period of time and in constant severity.

In 1957 Pfeffer, Stasior and Hinton[3] described a technique for inducing pancreatitis in dogs by creating an isolated loop of duodenum into which the pancreatic ducts drain, and then restoring gastrointestinal continuity by a gastroduodenal anastomosis. The common bile duct is ligated to exclude bile as a factor. As duodenal-loop distension develops, changes occur in the pancreas, beginning with the appearance of edema at four hours and progressing to hemorrhagic necrosis by 11 hr. Pfeffer believed that these changes resulted from vascular occlusion secondary to distension of the duodenal loop. More recently other workers[4, 5] have shown that reflux of duodenal contents into the pancreatic duct occurs in the Pfeffer model and that when the pancreatic ducts are ligated and reflux prevented, pancreatitis does not develop. However, in our hands, Pfeffer's method proved unreliable.

We originally believed that if ligation of the superior pancreaticoduodenal vein was added to this procedure, pancreatitis would occur that was constant in severity and rate of development.[6] With more experience this also proved not to be the case. Approximately 70% of the dogs developed severe pancreatitis, the others showing little change or merely some edema.

In an attempt to induce pancreatitis that would be constant in severity and rate of development, the Pfeffer method was modified by "splinting" the papilla of the inferior pancreatic duct to provide an unobstructed pathway between the duodenum and the intrapancreatic ductal system.

METHODS

Surgery

The technique for inducing pancreatitis is shown in Fig. 1. Mongrel dogs weighing from 10 to 24 kg. were used. The animals were fasted for 24 hr. preoperatively and an intravenous infusion of 5% dextrose in water was administered during the experi-

*From the Surgical-Medical Research Institute, University of Alberta, Edmonton, Alta.

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‡Edmonton Civic Employees Association Research Fellow.

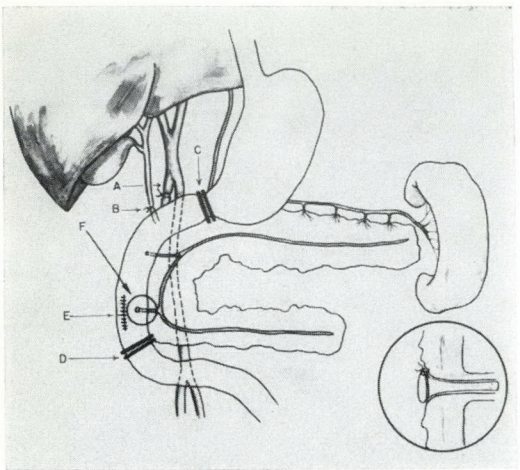

Fig. 1.—Technique for inducing pancreatitis. Ligatures are placed on the superior pancreaticoduodenal vein (A) and the common bile duct (B). Clamps are placed across the duodenum at the pylorus (C) and distal to where the pancreas ends in the dorsal mesentery (D). The duodenum is opened along the antimesenteric border (E) and a polyethylene cannula is inserted into the pancreatic duct (F). A magnified view of the entrance of the inferior pancreatic duct into the duodenum with the cannula in place is shown in the lower right-hand corner.

ment. A midline upper abdominal incision was used. Upon entering the peritoneal cavity, the common bile duct and the superior pancreaticoduodenal vein were ligated. Two crushing clamps were placed across the duodenum, one at the pylorus, and the other immediately distal to where the pancreas ends in the mesentery of the duodenum. This procedure creates a closed loop into which the pancreatic ducts drain.

The closed loop was then opened along the antimesenteric border and the papilla of the inferior pancreatic duct identified. A 7-mm. length of No. 160 polyethylene tubing with a flanged end was placed in the pancreatic duct and held there with a suture. A free flow of pancratic juice through the cannula was observed. The duodenal loop was then carefully irrigated with 50 ml. of sterile saline and the opening in the duodenum was closed. The abdominal incision was covered with a drape allowing the pancreas to be examined at half-hour intervals and the gross changes were recorded.

At the time of sacrifice, specimens of pancreas were taken from each animal for histologic examination. They were fixed in formalin and assessed microscopically; during this assessment, attention was focused on two factors: the degree of hemorrhagic necrosis and the degree of inflammatory cell infiltration.

TABLE I.—Grading of Hemorrhagic Necrosis Observed Microscopically in the Pancreas

Grade	*Microscopic findings*
I.	Edema
II.	Scattered areas of hemorrhagic necrosis
III.	Extensive hemorrhagic necrosis
IV.	Complete destruction of pancreas

The degree of hemorrhagic necrosis observed microscopically was graded in the manner shown in Table I. Four grades of change were observed ranging from edema only, to hemorrhagic necrosis of the entire pancreas.

The degree of inflammatory cell infiltration observed microscopically was graded in the manner shown in Table II. Here again, four grades of change were observed ranging from negligible, to severe infiltration with microabscess formation.

TABLE II.—Grading of Inflammatory Cell Infiltration Observed Microscopically in the Pancreas

Grade	*Microscopic findings*
A	Negligible
B	Minimal
C	Moderate
D	Severe, microabscess formation

Control Series

The technique described for inducing pancreatitis was carried out on 10 dogs. The pancreas was examined at half-hour intervals and the gross changes were recorded. All 10 dogs were sacrified six hours after surgery and specimens were taken for histologic examination.

Trasylol-treated Series

In a second series of 10 dogs, the technique for inducing pancreatitis was carried out in a manner identical to the control series. However, just before closure of the duodenum, 75,000 units of the polypeptide, Trasylol, (FBA Pharmaceuticals Ltd.) was introduced into the duodenal loop. These

animals were observed in the same manner as those in the control series and all were sacrificed six hours after surgery.

Determination of Proteolytic Activity in Pancreatic Edema Fluid

In a third series of six dogs, an attempt was made to study the effect of duodenal reflux on the release of proteolytic enzymes in the interstitial tissue of the pancreas. Pancreatitis was induced in the same manner as in the control series. As pancreatic edema developed, 0.1-ml. samples of the edema fluid were aspirated using a No. 27 gauge needle.[7] Specimens were taken at half-hour intervals and the level of proteolytic activity in each specimen was determined. Proteolytic activity was determined by the method of Blackwood and Mandl[8] using benzoyl arginine-β-naphthylamide as the substrate.

Results

Control Series

In all 10 dogs the gross changes observed in the pancreas began with the appearance of edema by one hour. Between two and four hours, the edema increased and petechial hemorrhages began to appear in the pancreatic parenchyma. Following this the degree of hemorrhage gradually increased so that, at six hours, the pancreas had become a hemorrhagic mass.

The degrees of hemorrhagic necrosis and inflammatory cell infiltration observed microscopically are shown in Table III. It is apparent from these results that extensive hemorrhagic necrosis developed in all animals within six hours. The degree of inflammatory cell infiltration was variable in that all grades were observed. Only two animals showed negligible infiltration.

TABLE III.—Control Series. Microscopic Findings in the Pancreas at Six Hours

Degree of hemorrhagic necrosis		*Degree of inflammatory cell infiltration*	
Grade	*No. of dogs*	*Grade*	*No. of dogs*
I	0	A	2
II	0	B	3
III	10	C	3
IV	0	D	2

Trasylol-treated Series

Grossly, in the 10 dogs treated with Trasylol, varying degrees of edema appeared in the pancreas within the first two hours. Except for one animal that developed a few petechiae in the pancreas, there was no progression of the process and edema was the only finding at the time of sacrifice.

The degrees of hemorrhagic necrosis and inflammatory cell infiltration observed microscopically are shown in Table IV. The degree of hemorrhagic necrosis is significantly different from that seen in the control series. One animal developed a few scattered areas of hemorrhagic necrosis while, in the other nine, edema was the only finding. All degrees of inflammatory cell infiltration were seen, as in the control series. Three animals in this series had negligible infiltration.

TABLE IV.—Trasylol-treated Series. Microscopic Findings in the Pancreas at Six Hours

Degree of hemorrhagic necrosis		*Degree of inflammatory cell infiltration*	
Grade	*No. of dogs*	*Grade*	*No. of dogs*
I	9	A	3
II	1	B	4
III	0	C	2
IV	0	D	1

Determination of Proteolytic Activity in Pancreatic Edema Fluid

The results of this study are shown in Fig. 2. It is apparent that the level of pro-

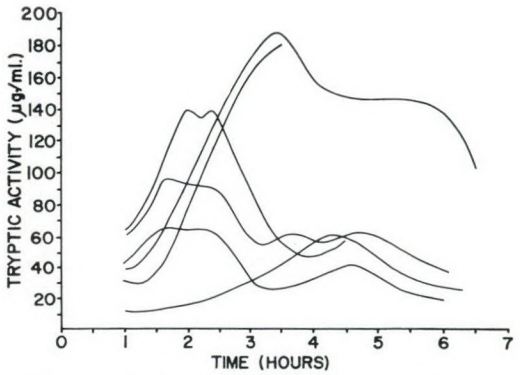

Fig. 2.—Graph of tryptic activity in pancreatic edema fluids during the development of severe pancreatitis.

teolytic activity in pancreatic edema fluid during the development of pancreatitis begins to rise in the early stages, reaches a plateau and then falls off as the process becomes more advanced.

Discussion

Difficulties in producing pancreatitis by the Pfeffer method have been reported previously.[9] There is little doubt that the mechanism of development depends upon reflux of the duodenal contents into the pancreatic duct.[4, 5] Inconsistent production of pancreatitis by the Pfeffer method may be due to some unknown factor which prevents reflux. In some dogs, the intraduodenal portion of the pancreatic duct was more oblique and longer than in others, possibly producing a more competent sphincter mechanism. In a few dogs, the distal line of transection of the duodenum was found to be so close to the papilla of the inferior pancreatic duct that closure of the bowel and the turning-in of mucosa obstructed the duct. By splinting the papilla of the inferior pancreatic duct, any factor that tends to prevent reflux is overcome. Also identifying the papilla of the inferior pancreatic duct each time and not transecting the duodenum, obviates the problem of obstruction of the pancreatic duct in the distal suture line. This procedure is technically easier than the Pfeffer method because the increased efficiency of reflux hastens the development of the process.

Trasylol is derived from bovine parotid glands. The chief action of this substance is inactivation of proteolytic enzymes, including kallikrein, trypsin and chymotrypsin at a pH of 5.0 to 7.8. One KI unit (kallikrein inhibitor unit) of Trasylol is said to inactivate two micrograms of trypsin by 95%. When Trasylol was instilled into the duodenal loop at the time of surgery, inflammatory cell infiltration still occurred, as in the contol group. However, hemorrhagic necrosis of the pancreas was minimal. This suggests that Trasylol inhibited the release of pancreatic proteolytic enzymes suppressing the development of hemorrhagic necrosis.

Studies on proteolytic activity in pancreatic edema fluid during the development of pancreatitis indicate that proteolytic enzymes are activated before the development of hemorrhagic necrosis.

These findings lend support to the belief that trypsin, which is the most plentiful pancreatic proteolytic enzyme, plays the major role in the development of acute hemorrhagic pancreatitis.

Summary

A satisfactory method of inducing pancreatitis of constant severity and rate of development in the dog is described. The method is a modification of the technique described by Pfeffer. A closed duodenal-loop obstruction is created and the inferior pancreatic duct papilla is splinted so that reflux of duodenal contents into the pancreas is assured. Severe hemorrhagic pancreatitis develops over a period of six hours.

When the antitryptic agent, Trasylol, was placed in the obstructed duodenal loop, inflammation occurred, but hemorrhagic necrosis did not develop.

A study of proteolytic activity in pancreatic edema fluid during the development of pancreatitis demonstrates that proteolytic activity increases as pancreatitis becomes severe and then falls once necrosis is evident.

The authors wish to thank FBA Pharmaceuticals Ltd. for supplying the Trasylol used in this study.

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Résumé

L'article décrit une méthode satisfaisante pour provoquer chez le chien une pancréatite d'une gravité et d'une vitesse d'évolution constantes. Cette méthode est une modification de celle déjà exposée par Pfeffer. On crée une occlusion d'une anse duodénale fermée et la papille du canal de Wirsung inférieure est éclissée de façon à assurer le reflux du contenu duodénal. Une pancréatite hémorragique grave se développe sur une période de six heures.

Une fois le médicament antitryptique instillé dans l'anse duodénale obstruée, l'inflammation s'est produite, mais la nécrose hémorragique ne s'est pas produite.

L'étude de l'activité protéolytique du liquide d'œdème pancréatique pendant l'évolution de la pancréatite démontre que l'activité protéolytique augmente à mesure que la pancréatite devient plus grave puis tombe dès que la nécrose est évidente.

THE EFFECT OF HYPERBARIC OXYGEN ON L-NORADRENALINE-INDUCED MYOCARDIAL NECROSIS*†

FRANCISCO VIOLAGO, JR., M.D. and ISRAEL PENN, M.B., B.Ch., F.R.C.S.[Eng. & C],
Winnipeg, Man.

Exposure of an animal or a patient to 100% oxygen at increased pressure results in a considerable elevation of the oxygen tension and content of the arterial blood.[1] Hyperbaric oxygen has been used in the treatment of a wide variety of anoxic states, either those involving the body as a whole or those confined to certain organs or tissues. Among the conditions that have been treated with hyperbaric oxygen are myocardial ischemia and infarction. Two methods for producing acute myocardial lesions in experimental animals have been used—ligation of a branch of one of the coronary arteries or the injection of microspheres into the coronary circulation. After the arterial occlusion had been produced, the animals were treated with hyperbaric oxygen. Conflicting results have been reported, some[2-12] claiming that hyperbaric oxygen therapy is beneficial while others[13-15] have stated that it is of no value.

In an attempt to shed further light on this problem, we investigated the effect of hyperbaric oxygen upon a myocardial ischemic process produced relatively slowly by prolonged intravenous infusion of L-noradrenaline.

Materials and Methods

Thirty adult mongrel dogs, weighing between 10 and 18 kg., were studied. Each animal was anesthetized with 30 mg./kg. of pentobarbital sodium. An endotracheal tube was passed to ensure a free airway. Both femoral arteries and one femoral vein were cannulated. One femoral artery was connected *via* a Statham pressure transducer to a Grass polygraph and arterial blood pressure was recorded at 15-min. intervals. The other arterial cannula was used for collecting samples for measurement of hematocrit, viscosity, plasma proteins, serum glutamic oxaloacetic transaminase (SGOT), pO_2, pCO_2 and pH. A Brookfield viscometer* was used for measurements of viscosity. A Radiometer monitor† was used to measure blood pH, pCO_2 and pO_2 levels at atmospheric pressure, while a Beckman gas analyzer‡ was used to measure blood pO_2 levels at pressures higher than atmospheric.

*From the Department of Surgery, The University of Manitoba, Winnipeg, Man.
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*Synchrolectic Viscometer, Model LVT; Brookfield Engineering Laboratories, Stoughton, Mass.
†Radiometer, Model PHA-927 gas monitor; Radiometer Co., Copenhagen, Denmark.
‡Beckman Physiological Gas Analyzer, Model No. 160; Beckman Instruments Inc., Palo Alto, Calif.

Electrocardiographic tracings (Leads I, II, III, AVL, AVR and AVF) were made at 15-min. intervals.

Each animal was infused with L-noradrenaline at a rate of 2 μg./kg./min. for a period of five hours. Fifty millilitres of 5% dextrose in water were used as a diluent for the L-noradrenaline. This solution was injected into the femoral vein cannula by means of a Harvard infusion pump.*

The animals were divided into two groups. One group breathed room air at one atmosphere absolute pressure and served as a control. The second group breathed 100% oxygen and was pressurized to two atmospheres absolute. Pressurization began 15 min. before the infusion of L-noradrenaline and ended 15 min. after the infusion of L-noradrenaline. The animals were thus exposed to hyperbaric oxygen for a period of 5½ hr.

The dogs were sacrificed 30 min. after the infusion of L-noradrenaline had been completed. Full autopsies were performed in all animals. The heart was removed and its epicardial surface was carefully inspected for areas of hemorrhagic necrosis. The chambers were then opened and similarly examined.

The extent of the lesions was measured in the following manner: The left ventricle, which was the site of the great majority of the lesions, was opened widely and laid out flat. A colour transparency of the endocardial surface was made and projected upon a small screen. Tracings of the outlines of the endocardial surface of the ventricle and of all foci of hemorrhagic necrosis were made. Using a planimeter, the surface area of each of the necrotic foci and of the endocardial surface of the ventricle were mea-

TABLE I.—Changes in Blood Pressure and Electrocardiogram in Control Animals

	Blood pressure							*ECG*	
Dog number	*Initial BP*	*Peak BP*	*Time at which peak BP reached (min.)*	*BP at 3 hr.*	*BP at 5 hr.*	*BP before sacrifice*	*Heart rate and rhythm*	*Q waves*	*ST changes*
1	120/115	380/150	3	145/105	150/115	135/75	Ventricular extrasystoles	Present in Leads II, III and AVF	Elevated in Leads II, III and AVF
2	175/135	335/190	3	150/110	120/85	70/50	Ventricular extrasystoles	Present in Leads II, III and AVF	Elevated in Leads II, III and AVF
3	150/100	320/185	1	150/90	140/80	125/75	Unaltered	Present in Leads II, III and AVF	Elevated in Leads II, III and AVF
4	130/105	360/270	13	170/135	150/130	135/105	Unaltered	Absent	Absent
5	200/130	385/225	1	170/120	140/110	130/90	Unaltered	Absent	Depressed in Leads II, III and AVF
6	160/115	230/145	2	160/110	120/80	60/40	Bradycardia and ventricular extrasystoles	Present in Leads II, III and AVF	Elevated in Leads II, III and AVF
7	120/95	350/230	1	165/115	110/80	60/45	Ventricular extrasystoles	Absent	Absent
8	185/125	370/270	2	115/80	80/50	60/40	Unaltered	Present in Leads II, III and AVF	Elevated in Leads II, III and AVF
9	170/110	400/240	1	190/125	180/115	120/80	Unaltered	Absent	Absent
10	210/120	360/140	5	140/110	140/100	110/90	Unaltered	Absent	Absent
11	170/110	280/150	15	195/130	180/135	150/100	Unaltered	Absent	Absent
12	160/105	370/150	15	190/115	105/80	105/80	Bradycardia and ventricular extrasystoles	Absent	Absent
13	190/145	320/150	10	180/115	180/115	120/75	Unaltered	Present in Leads II, III and AVF	Depressed in Leads II, III and AVF
14	140/95	180/130	115	165/105	165/105	125/90	Ventricular extrasystoles	Absent	Absent
15	130/50	275/125	16	180/90	180/90	110/60	Unaltered	Absent	Elevated in Leads II, III and AVF

*Harvard Dual Reciprocal Infusion/Withdrawal Pump, Model No. 600-920; Harvard Apparatus Co. Inc., Dover, Mass.

TABLE II.—Changes in Blood Pressure and Electrocardiogram in Hyperbaric Animals

	Blood pressure						ECG		
Dog number	Initial BP	Peak BP	Time at which peak BP reached (min.)	BP at 3 hr.	BP at 5 hr.	BP before sacrifice	Heart rate and rhythm	Q waves	ST changes
16	120/80	210/135	12	190/110	165/75	150/75	Bradycardia	Absent	Absent
17	130/80	235/130	10	190/160	190/110	120/100	Unaltered	Absent	Absent
18	160/110	240/135	10	220/160	210/116	140/90	Bradycardia	Absent	Absent
19	130/80	270/160	8	115/70	135/90	90/50	Bradycardia	Absent	Absent
20	195/125	235/135	17	180/120	200/130	140/85	Unaltered	Absent	Absent
21	165/115	335/195	1	140/100	150/100	120/85	Ventricular extrasystoles	Absent	Absent
22	110/75	320/190	1	165/115	170/120	95/70	Unaltered	Absent	Absent
23	175/100	320/210	1	175/125	195/120	140/95	Unaltered	Absent	Depressed in Leads II, III and AVF
24	120/75	380/100	1	250/190	170/110	135/95	Unaltered	Absent	Absent
25	160/125	250/145	18	150/110	160/130	130/110	Unaltered	Absent	Absent
26	180/140	310/130	50	150/130	155/95	140/70	Unaltered	Absent	Absent
27	130/110	220/130	29	150/100	180/120	65/25	Unaltered	Absent	Absent
28	180/130	240/150	28	185/115	220/135	175/110	Unaltered	Absent	Depressed in Leads II, III and AVF
29	160/140	215/175	30	175/140	205/150	190/140	Unaltered	Absent	Absent
30	195/135	320/150	40	270/165	250/160	185/135	Bradycardia & ventricular extrasystoles	Absent	Depressed in Leads II, III and AVF

sured. Each reading was done three times and a mean of the three readings was used to express the surface area of the site being measured. The sum of the surface areas of the necrotic foci was expressed as a percentage of the surface area of the endocardial surface of the ventricle.

Histologic studies were made of the heart, lungs, liver, spleen, kidneys, stomach, intestines, pancreas and adrenal glands.

Results

Blood Pressure and Electrocardiographic Changes (Tables I and II)

The blood pressure became elevated shortly after the L-noradrenaline was administered, and reached a peak within one to 50 min. after the infusion started. The blood pressure then declined, and in most of the animals was at a lower level at the time of sacrifice than it had been at the beginning of the experiment. Seven animals showed a marked fall in blood pressure after the L-noradrenaline infusion was terminated. There was no correlation between the fall in blood pressure and the development of lesions in the myocardium. There was a fairly close correlation between the development of electrocardiographic changes in Leads II, III and AVF and the development of extensive lesions in the myocardium. With one exception (Dog 14), animals with lesions involving more than 10% of the endocardial surface of the left ventricle showed electrocardiographic changes.

Changes in the Blood (Table III)

SGOT

Slight elevation of the SGOT occurred in seven of the control animals and in seven of the treated animals. In the control animals, the SGOT rose from a mean of 24.4 $\pm$ 3.5 at the beginning of the experiment to 56.0 $\pm$ 8.2 at the end of the experiment. In the treated animals, the SGOT rose from a mean of 21.8 $\pm$ 2.0 to 42.4 $\pm$ 4.3.

Hematocrit, Plasma Proteins and Viscosity

In the majority of animals, the hematocrit and blood viscosity increased during the course of the experiment. In the control animals the mean hematocrit rose from 45.6 $\pm$1.5 at the beginning of the experiment to

TABLE III.—Blood Changes

Parameter measured	Time of measurement*	Control animals		Hyperbaric animals	
		Mean	Standard error	Mean	Standard error
SGOT (units/100 ml.)	B	24.4	±3.5	21.8	±2.0
	E	56.0	±8.2	42.4	±4.3
Hematocrit (%)	B	45.6	±1.5	40.4	±1.6
	E	57.3	±2.8	47.1	±1.5
Plasma proteins (mg./100 ml.)	B	6.15	±0.21	5.55	±0.35
	E	6.30	±0.25	6.27	±0.11
Blood viscosity (centipoise at 60 rpm)	B	4.27	±0.13	3.83	±0.15
	E	6.56	±0.73	4.17	±0.15

*B—Measurements made at the beginning of the experiment.
E—Measurements made at the end of the experiment.

57.3 ± 2.8 at the time of sacrifice. In the treated animals, the rise was from 40.4 ±1.6 to 47.1 ± 1.5 at the time of sacrifice.

In the control animals, the blood viscosity increased from 4.27 ± 0.13 centipoise at 60 rpm at the beginning to 6.56 ± 0.73 at the time of sacrifice. The increase in the treated animals was from 3.83 ± 0.15 to 4.17 ± 0.15.

In many of the animals there was also a slight increase in the serum proteins but this was not as consistent as the increase in hematocrit and viscosity. In the control animals, the serum proteins rose from 6.15 ± 0.21 mg.% at the beginning of the experiment to 6.30 ± 0.25 at the time of sacrifice. In the treated animals, the increase was from 5.55 ± 0.35 to 6.27 ± 0.11.

TABLE IV.—Extent of Myocardial Lesions

Control animals		Hyperbaric animals	
Dog number	Area of infarcts* (%)	Dog number	Area of infarcts* (%)
1	7.9	16	0.0
2	100.0	17	4.8
3	59.0	18	5.4
4	6.9	19	3.5
5	9.6	20	10.3
6	14.7	21	4.7
7	0.6	22	0.0
8	35.6	23	24.2
9	8.1	24	2.6
10	9.9	25	0.0
11	8.2	26	0.0
12	9.2	27	0.0
13	31.3	28	33.8
14	20.5	29	0.0
15	37.4	30	9.9

*Area of infarction expressed as a percentage of the endocardial surface of the left ventricle.

pO_2, pCO_2 and pH

The majority of the pO_2 measurements in the control animals were in the range between 70 and 100 mm.Hg. On occasion the pO_2 fell below the former level, following the administration of maintenance doses of pentobarbital sodium. Similar findings were obtained in the treated animals before exposure to hyperbaric oxygen; while exposed to hyperbaric oxygen, the pO_2 rose to a level of between 908 and 1200 mm.Hg. When hyperbaric treatment was discontinued, the pO_2 fell to prepressurization levels. The pCO_2 and pH did not show any consistent changes.

Gross Changes in the Heart (Table IV)

Focal areas of hemorrhagic necrosis were present in most of the animals. These were most consistently present on the endocardial surface of the left ventricle, with occasional hemorrhages on the endocardial aspect of the right ventricle or in the epicardium of both ventricles.

When present, the endocardial hemorrhages were situated in and immediately beneath the endocardium and did not appear to extend to any depth into the myocardium. These hemorrhagic areas were

present in all the control animals but were absent in six of the 15 treated animals.

The area of the endocardial surface of the left ventricle involved by hemorrhagic necrosis was 23.9% (S.E. ± 6.56) in the control animals, and 6.6% (S.E. ± 2.45) in the animals treated with hyperbaric oxygen. This difference is statistically significant ($P = 0.02$).

Histologic Changes in the Heart

Microscopic examination confirmed the presence of focal areas of subendocardial hemorrhage with disruption and necrosis of muscle fibres. In those animals in which no hemorrhagic areas were seen on gross examination, none were seen on miscroscopic examination.

Changes in Other Organs

Scattered hemorrhages were found in other organs, most commonly the lungs and the intestines. In both the control and treated groups, eight animals showed hemorrhages in the lungs; and seven in each group showed hemorrhages in the intestines. The hemorrhages, particularly those in the lungs, were more extensive in the animals treated with hyperbaric oxygen than in the control animals.

Occasional animals showed hemorrhages in the liver, kidneys or in the adrenal glands.

Discussion

Prolonged infusion of a large dose of L-noradrenaline in experimental animals has been shown to consistently produce focal areas of hemorrhagic necrosis in the heart, mainly subendocardial in distribution.[16-18] Raab[16] has shown that, when catecholamines are administered, oxygen uptake and utilization by some areas of the myocardium may be excessive, leaving adjacent areas with inadequate supplies of oxygen so that focal necroses develop. The inner layers of the ventricles in particular are at a disadvantage, due to compression of their vessels when the intraventricular pressure rises.

Another factor that may contribute to the development of anoxia is increased viscosity of the blood, which may hamper adequate tissue perfusion. Finnerty, Buchholz and Guillaudeu[19] have demonstrated that L-noradrenaline administration causes a decrease in the plasma volume. The present studies indicate that a rise in the hematocrit and, to a lesser degree, a rise in the plasma proteins occurs, with a resultant increase in the blood viscosity.

There is no simple way of quantitating the extent of myocardial necrosis.[20] As the foci of hemorrhagic necrosis in the present experiments were very superficial and chiefly subendocardial in distribution, we used the method described above to measure the portion of the surface area of the endocardium that underwent necrosis.

The results in the present study indicate that hyperbaric oxygen at a pressure of two atmospheres absolute does exert a beneficial effect upon L-noradrenaline-induced myocardial necrosis. Six of 15 treated animals had no lesions whatever, whereas all the control animals had lesions. The lesions in the control animals involved 23.9% of the endocardial surface of the left ventricle whereas only 6.6% of this surface was involved in the treated animals. These findings are in keeping with the results of other workers.[2, 3, 5-12]

No striking changes in the SGOT levels were seen in the present studies, a finding similar to that of Harris and Hitchcock.[21] This may be explained by the fact that the measurements were made relatively early in the course of infarction. After a myocardial infarction, the rise in SGOT levels is usually fairly slow and takes 24 hr. to reach a peak.[20]

The more extensive hemorrhages in the lungs noted in the treated animals as compared with the controls is not surprising in view of the report by Bean and Johnson[22] that adrenaline considerably enhances the toxic effects on the lungs of oxygen at high pressure. Presumably, L-noradrenaline has a similar action.

We may, therefore, conclude that while hyperbaric oxygen therapy has a protective effect and reduces the incidence and extent of necrosis produced in the myocardium by L-noradrenaline, its toxic effects on the lungs may be enhanced by the simultaneous administration of L-noradrenaline.

SUMMARY

Focal areas of myocardial infarction were produced in dogs by the prolonged infusion of L-noradrenaline.

Two groups of animals were studied. A control group that received no treatment and a group that were treated with hyperbaric oxygen before, during and after the infusion of L-noradrenaline.

Hyperbaric oxygen exerts a significant protective effect upon the myocardium exposed to ischemia.

L-noradrenaline, however, potentiates the toxic effects of hyperbaric oxygen upon the lungs.

We wish to thank Dr. C. W. Birkett of Winthrop Laboratories, Aurora, Ont. for kindly supplying the L-noradrenaline used in these studies. We are also grateful to Dr. Drummond Bowden of the Department of Pathology, The University of Manitoba, Winnipeg, Man. for his invaluable help with the pathologic studies.

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Résumé

On a provoqué des zônes focales d'ischémie myocardique chez 30 chiens bâtards adultes. Les chiens ont été séparés en deux groupes. Un groupe, servant de témoins, n'a pas reçu de traitement. Le groupe expérimental a été traité à l'oxygène hyperbarique à la pression absolue de deux atmosphères, pendant et après la perfusion de L-noradrénaline, durant une période totale de 5½ heures pour chaque animal.

On a constaté que l'oxygénation hyperbarique exerce une action protectrice notable sur l'ischémie du myocarde. Cependant, la L-noradrénaline a potentialisé les effets toxiques de l'oxygène hyperbarique sur les poumons.

EFFECTS OF CORTISONE AND AN ANABOLIC ANDROGEN ON THE FRACTURED HUMERUS IN GUINEA PIGS: CLINICAL AND HISTOLOGICAL STUDY OVER A SIX-WEEK PERIOD OF FRACTURE HEALING*

H. MURAKAMI, M.D., Ph.D.† and K. KOWALEWSKI, M.D., Ph.D.,
Edmonton, Alta.

Steroid hormones are known to affect mesenchymal tissue and influence growth and development of cartilage and bone.[1, 2] The inhibitory action of anti-anabolic corticoids is explained by its action on fibroblasts,[3] hyaluronate,[4] sulphated polysaccharides[2, 5-7] synthesis of matrix proteins, fibrinogenesis[1, 2] and bone minerals.[2] There is considerable experimental and clinical evidence that some anabolic steroids have an opposite action to cortisone and may prevent the inhibitory effects of corticoids on bone growth, formation and healing.[2, 6, 8, 9]

Retardation of fracture healing in cortisone-treated animals was first reported by Ragan *et al.*[10] We previously studied the various effects of both cortisone and anabolizers on growing bones in rodents and birds,[2, 6, 8] and on healing of fractures in rats. We were able to show that the inhibitory action of cortisone on various elements of callus and on the healing process may be prevented or counteracted by some anabolic androgens, which also promote bone healing under certain experimental conditions.[11-17]

These findings, confirmed by others,[18] were mostly concerned with the composition of callus or its tensile strength in the early stages of fracture healing. It was not, however, clearly demonstrated whether actual delay or acceleration of the healing process and of the final fracture union does occur. From the practical point of view, this final result of steroid action on bone healing appears to be most important to clinicians. Existing experimental data on fracture healing in steroid-treated animals is apparently not sufficiently convincing to encourage the clinical use of anabolizers. There is still a need for a detailed study of fracture healing during the several weeks after fracture in normal and steroid-treated animals. The present investigation was an attempt to clarify further the problem of steroid action on fracture healing by both clinical and histological methods.

Methods

Male guinea pigs with an average body weight of 550 g. used in this experiment, were subjected to a closed complete fracture of the left humerus. This was performed manually under light ether anesthesia. Animals were kept in separate cages and the fractures were left to heal unsupported.

Cortisone acetate (Cortone, Merck) was injected subcutaneously in a dose of 5 mg./kg. body weight daily, beginning two weeks before fracture and continuing through the experiment. Anabolic steroid 17β-hydroxy-17-α-methylandrostano [3, 2-c]

*From the Surgical-Medical Research Institute, University of Alberta, Edmonton, Alta.
†Fellow in Experimental Surgery.

pyrazole (Winstrol; Winthrop Lab.) a depot-injectable preparation was given twice, each time in a dose of 50 mg./kg. body weight subcutaneously; the first dose two weeks before, and the second dose two weeks after the fracture.

The animals were divided into four groups: (1) No treatment; (2) treated with cortisone; (3) treated with Winstrol; and (4) treated with both steroids. The guinea pigs were sacrificed in small groups on the fourth day and at the end of the first, second, third, fourth, fifth and sixth weeks after fracture. There were three animals in each group; this permitted a satisfactory study of each stage in bone healing.

After radiologic examination, the humeri were carefully dissected and macroscopic findings recorded. The fractured bone with a thin layer of muscle was then fixed in 10% neutral formalin, decalcified with 5% nitric acid, and paraffin blocks prepared. Serial sections (6μ) were then cut. Hematoxylin-eosin, toluidin-blue and alcian-blue stains were used for preparation of the histological slides.

Results

Evidence of the clinical union of fractured humeri (Table I), supported by radiological observations (Table II) demonstrated the delaying action of cortisone and the promoting action of an anabolizer. The effects of these hormones are evident at the early stages of healing but are no longer detected in the last two weeks of the experiment.

TABLE I.—Effect of Steroids on Clinical Union of Fractured Humerus in Guinea Pigs

		Weeks after fracture				
Group	*Treatment*	*2*	*3*	*4*	*5*	*6*
1	None	*	+	+	++	++
2	Cortisone	0	0	+	++	++
3	Anabolic steroid**	*	+	++	++	++
4	Both steroids	0	*	+	++	++

0 = No union
* = Limitation of movement
\+ = Almost united
++ = Complete clinical union
** = Winstrol

TABLE II.—Effects of Steroids on Radiological Union of Fractured Humerus in Guinea Pigs

		Weeks after fracture				
Group	*Treatment*	*2*	*3*	*4*	*5*	*6*
1	Control	*	*	++	+++	+++
2	Cortisone	*	*	*	++	+++
3	Anabolic steroid**	*	++	++	++	+++
4	Both steroids	*	*	++	++	+++

* = Periosteal thickening
++ = Bridging of callus
+++ = Radiological union
** = Winstrol

Bone Healing in the Normal Animal

Histology.—In order to evaluate the hormonal effects on fracture healing, the normal pattern of healing in the guinea pig will be presented in detail. Generally, we observed three types of bone formation: (a) Osteogenic cell proliferation and direct bone formation under the intact periosteum, including the vicinity of the fracture site. (b) Fibrous proliferation with cartilage formation and replacement of cartilage by endochondral bone at the disrupted periosteum, mainly at the fracture site. (c) Direct endosteal bone formation in the marrow space.

These three types of bone formation were notable during the six-week period of fracture healing, under the conditions of this experiment. A detailed study of fracture healing in normal, untreated guinea pigs, performed on the fourth day and at the end of the first, second, third, fourth, fifth and sixth weeks after fracture showed:

The fourth day of fracture.—Extensive osteogenic-cell proliferation under the intact periosteum; osteoblasts had already differentiated and a small amount of osteoid was present just outside the fractured cortical bone; endosteal bone formation was initiated. Between the fractured fragments, a small amount of hematoma was still present and fibrin networks were forming; fibrous proliferation appeared around the fractured bone and in the deep layer of the muscles; no cartilage was observed.

First week of fracture.—The osteogenic-cell layer under the intact periosteum showed marked increase in thickness, and contained numerous osteogenic cells and newly formed cancellous bone; newly formed bone trabeculae occupied about 70% of this layer and showed increasing maturity. The amount of endosteal bone increased at the fracture site and hematoma was almost resorbed; extensive fibrous tissue proliferation was noted around the fracture site; cartilage was found in the deep part of the fibrous tissue, close to

fractured cortical bone. Toluidin-blue stain showed metachromasia of cartilage matrix and alcian-blue stain demonstrated the presence of acid mucopolysaccharides in the cartilage matrix and adjacent fibrous tissues.

Second week of fracture.—Direct bone formation under the intact periosteum was almost terminated and proliferation of osteogenic cells ceased; the fibrous layer of the periosteum increased in thickness and newly formed cancellous bone increased in maturity. Endosteal bone formation was advanced but the trabeculae were still thin. At the fracture site, a further increase in fibrous tissue proliferation and cartilage formation was noted, but fibrous tissue was still dominant. Typical metachromasia was observed with toluidin-blue stain in cartilage matrix and in adjacent fibrous tissues. Alcian-blue stain showed marked increase of acid mucopolysaccharides in the cartilage and adjacent fibrous tissue. Endochondral bone formation was found, replacing the cartilage and starting from already directly formed subperiosteal and endosteal bone; near the ossification front, hypertrophy of chondrocytes became apparent, and numerous osteogenic cells and osteoblasts were found at the ossification front.

Third week of fracture.—Further maturation of subperiosteal new bone, which was taking on the appearance of cortical bone; fractured cortical bone showed progressive reorganization. At the fracture site, cartilage became a dominant structure and fibrous tissue, in reduced amounts, was noted at the periphery of the callus. Cartilaginous union became apparent. Typical metachromasia was still present and large amounts of acid mucopolysaccharides were detectable with alcian-blue stain. Slow but progressive endochondral bone formation was noted.

Fourth week of fracture.—Completion of maturation of subperiosteal new bone became apparent; this new bone replaced old fractured bone, now extensively reorganized and giving the appearance of cancellous bone. Cartilage was still a dominant structure at the fracture site, despite the marked progress in endochondral bone formation; endochondrally formed bone was still immature.

Fifth week of fracture.—Endochondrally formed bone became the dominant structure; the amount of cartilage was markedly reduced; no metachromasia was observed with toluidin-blue stain, and the amount of acid mucopolysaccharides, detectable with alcian-blue stain, appeared reduced.

Sixth week of fracture.—Reorganization of fractured cortical bone was completed and subperiosteal bone had replaced the old bone. The fracture gap was filled with endochondrally formed cancellous bone. Some of the bone trabeculae still contained cartilage components. The amount of cartilage appeared to be directly related to the condition of the periosteum after fracture; the disruption of the periosteum was more extensive and a larger amount of cartilage was formed.

Healing in Steroid-treated Animals

The action of steroids on bone healing.—It is already apparent from clinical and radiological studies that any effect steroids may have on bone healing was only evident in the first weeks after fracture. Many histological effects of steroids on bone growth and healing were previously described[2, 5, 9, 14] and it was decided that we would report the most striking differences between treated animals and controls, taking as the reference point, the histological pattern of the normal healing process described above.

First, in the cortisone group, hematoma at the fracture site was larger and persisted longer than in other groups: even in the fifth week of fracture, hematomas were found in the fibrous stroma at the fracture site.

Osteogenic cell proliferation.—On the fourth day and after one week of fracture, when the subperiosteal bone formation was very active, marked differences were observed between the experimental groups.

In the cortisone group, the number of osteogenic cells and osteoblasts was smaller than controls and this resulted in the formation of thinner and less mature trabeculae. In the Winstrol-treated group, quite the opposite effect was noted; the cell population was markedly larger than in controls and consequently thicker and more mature trabeculae were formed. In the

group treated with both steroids, the protective action of Winstrol was apparent because the cell population was larger than in controls. The results of cell counting illustrate this (Table III).

TABLE III.—The Number of Osteogenic Cells in Subperiosteal Regions of Comparable Fields and Identical Sizes†

Group	Treatment	Days after fracture 4	7	14
1	None	46	46	32
2	Cortisone	43	34+	29+
3	Anabolic steroid**	61*+	54*	35*
4	Both steroids	50	53	34
Significance		*P <0.1% 3rd *versus* 1st and 2nd groups +P <1% 3rd *versus* 4th group	*P <0.1% 3rd *versus* 1st and 2nd group +P <0.1% 2nd *versus* other groups	*P <5% 3rd *versus* 1st and 4th group +P <1% 2nd *versus* other groups

†At least 10 slides were studied in each group.
**Winstrol.

Between two and three weeks after fracture, when the subperiosteal bone formation was nearly terminated, the differences between the groups became less apparent and the subperiosteally formed bone showed a normal process of maturation.

Chondrocytes and endochondral bone formation.—In the cortisone-treated group, fibrous tissue proliferation at the fracture site was decreased in amount and delayed during the first two weeks of healing. Although the first cartilage appeared during the first week of fracture, the amount was very small; chondrocytes appeared immature, containing a large quantity of basophilic cytoplasm and large oval vesicular nuclei. Densely aggregated cells adjacent to the cartilage mass were similar to the ones described by Koekenberg[19] in the secondary ossification centres, and gave the clear impression that the differentiation of chondroblasts was retarded.

At the ossification front, associated with a marked reduction of osteogenic cells and osteoblasts, hypertrophied chondrocytes were absent; these cells still remained in the immature stage after two weeks of fracture. This delay in differentiation and maturation of chondrocytes in the cortisone-treated group improved after the third week and cartilage appeared normal after the fourth week.

Because of the smaller amount of cartilage formed in the cortisone group, the replacement of cartilage with bone progressed rapidly and bony union was, at the end of the experiment, almost identical with the control group.

Again the effect of the anabolizer was opposite to that observed in the cortisone-treated animals. The Winstrol-treated group showed a larger amount of cartilage formation and earlier appearance of well-matured chondrocytes, compared with the control group. Because of this larger amount of cartilage formation, some of the bones showed incomplete replacement of cartilage with endochondral bone in the fifth week after fracture and apparent delay of bony union.

In the Winstrol-cortisone group, the findings approached those of the control group, but there was still a tendency to greater than normal cartilage formation. Figures 1 to 12 illustrate some of the differences between the four groups, described above.

Stain for acid mucopolysaccharides.—These substances exhibit metachromasia with basic aniline dyes like toluidin-blue and give specific reaction with alcian-blue stain. The histochemical reactions determined with these stains give important information regarding the ground substance of bone.[12, 14] In normal fracture healing, a small amount of acid mucopolysaccharides appeared in the cartilage in the first week, and increased with the progressive growth of cartilage during the second and third weeks. After the fourth week, the amount of acid mucopolysaccharides decreased because the cartilage was progressively replaced by endochondral bone.

In the cortisone-treated group, the amount of acid mucopolysaccharides was reduced during the first three weeks as compared with other groups, but the differences were not apparent after the fourth week. In the Winstrol-treated group, much larger amounts of acid mucopolysaccharides were found, both in the early stage of healing and through all the stages examined. In the group treated with both steroids, the amount of acid mucopolysaccharides was larger, during all stages of healing, than in the control group. Direct relationship was noted between the intensity of alcian-blue reaction and the amount of cartilage present in healing fracture. Figures 13 and 14,

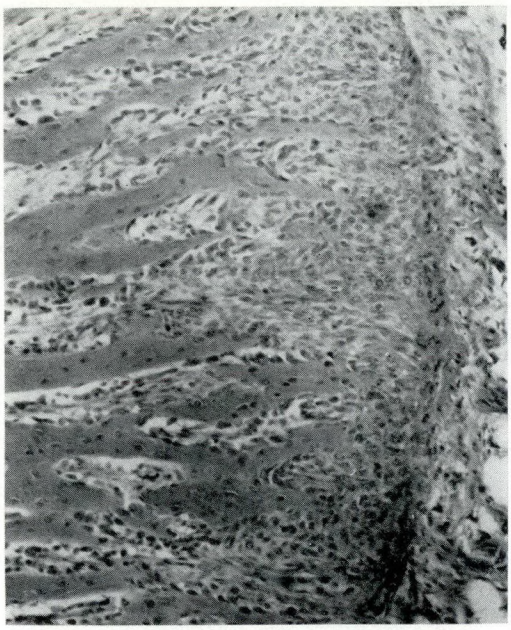

Fig. 1.—Control group, one week after fracture, showing marked proliferation of osteogenic cells. Osteoblasts have differentiated and cancellous bone has already formed. Trabeculae are still thin but numerous. Periosteum at the right. (H & E, original magnification x 125)

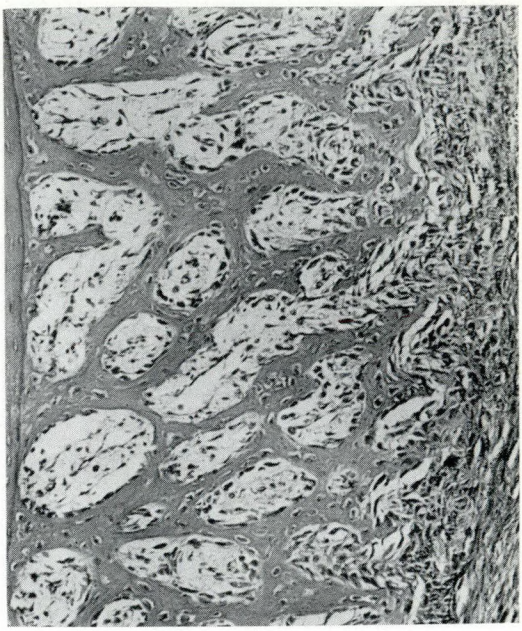

Fig. 2.—Cortisone group one week after fracture, showing reduced number of osteogenic cells and osteoblasts as compared with control group (Fig. 1). Trabeculae of new bone appear thin and immature. Periosteum at the right. (H & E x 125)

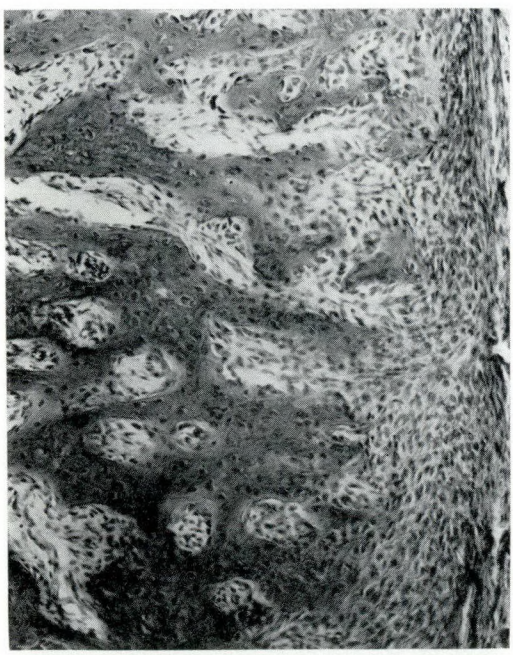

Fig. 3.—Winstrol group one week after fracture showing numerous osteogenic cells and osteoblasts with abundant intercellular substance. Trabeculae of newly formed cancellous bone are thicker and more mature than those of other groups (Figs. 1 and 2). Periosteum at the right. (H & E x 125)

Fig. 4.—Winstrol-cortisone group, one week after fracture shows numerous osteogenic cells and osteoblasts, thicker trabeculae and abundant intercellular substance. Picture comparable to control and Winstrol group. Periosteum at the right. (H & E x 125)

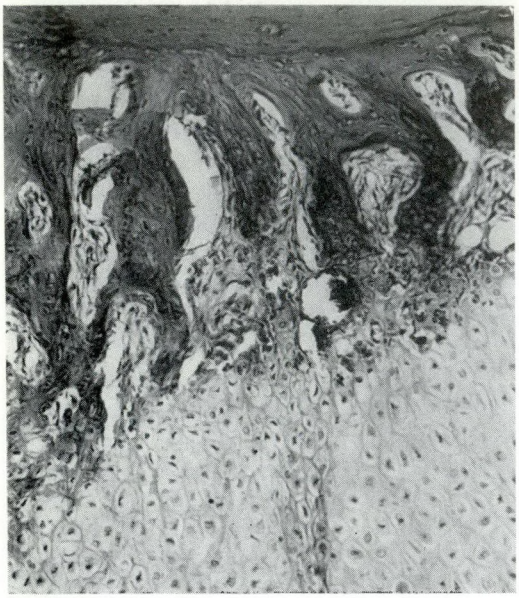

Fig. 5.—Control group two weeks after fracture. In the endochondral bone formation, well-matured hypertrophic chondrocytes are present near the ossification front. Numerous osteogenic cells and osteoblasts are seen at the ossification front, where endochondral bone is replacing cartilage. Fractured cortical bone at the top. (H & E x 125)

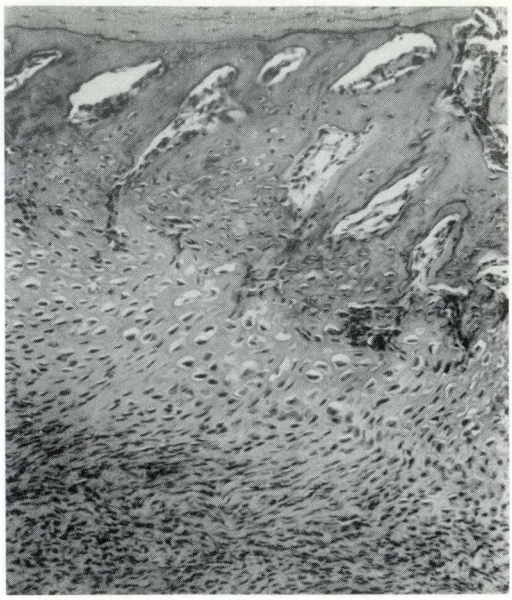

Fig. 6.—Cortisone group two weeks after fracture, showing immature appearance of chondrocytes and small amount of cartilage. A dense aggregation of cells at the periphery suggests that there is inhibition of chondroblast differentiation. No hypertrophied chondrocytes are noted. At the ossification front, the number of osteogenic cells and osteoblasts are reduced as compared with the control group. Some parts of the cartilage seem to be transformed directly into bone. Fractured cortical bone at the top. (H & E x 125)

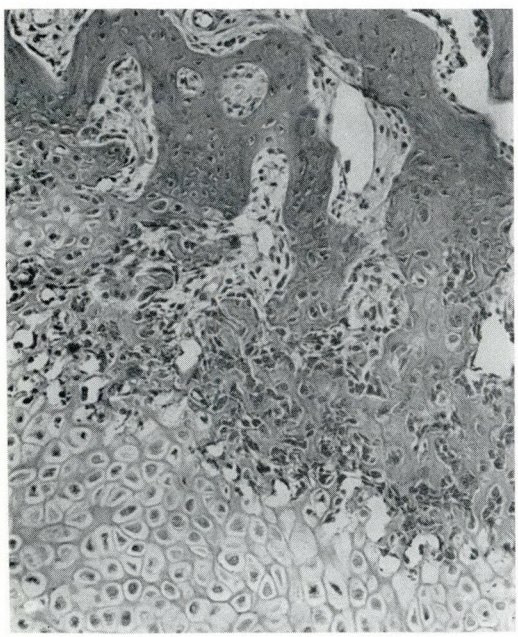

Fig. 7.—Winstrol group two weeks after fracture showing well-matured hypertrophied chondrocytes near the ossification front. Abundant intercellular substance between the chondrocytes is noted. A large number of osteogenic cells and osteoblasts can be seen at the ossification front. Fractured cortical bone at the top. (H & E x 125)

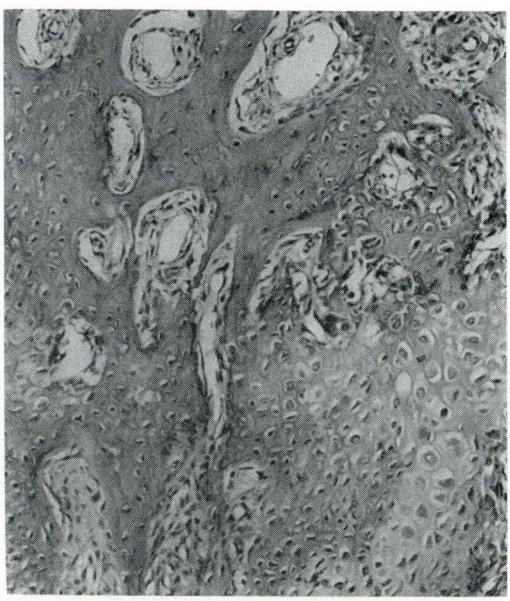

Fig. 8.—Winstrol-cortisone group two weeks after fracture showing well-matured and hypertrophied chondrocytes. Generally the picture appears similar to control and Winstrol group (Figs. 5 and 7). Fractured cortical bone at the top. (H & E x 125)

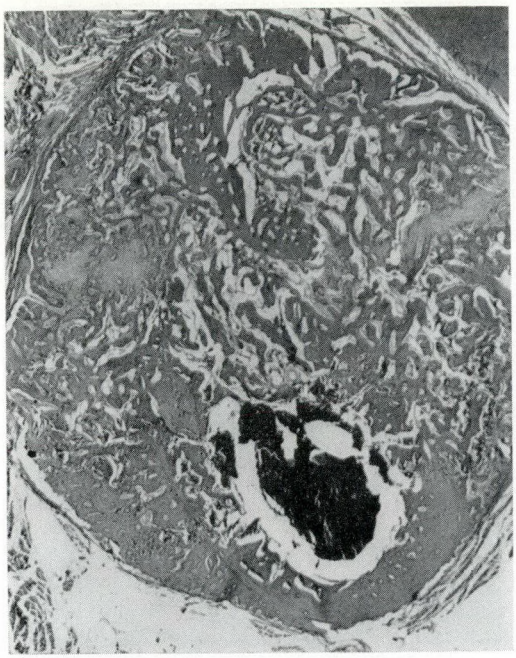

Fig. 9.—Control group five weeks after fracture. Callus has been filled with cancellous bone and bony union is present. A very small amount of cartilage can be seen at the periphery. (H & E, original magnification x 4)

Fig. 10.—Cortisone group five weeks after fracture showing callus filled with cancellous bone, and bony union comparable to the control group (Fig. 9). Cartilage is present in small amount at the periphery. (H & E x 4)

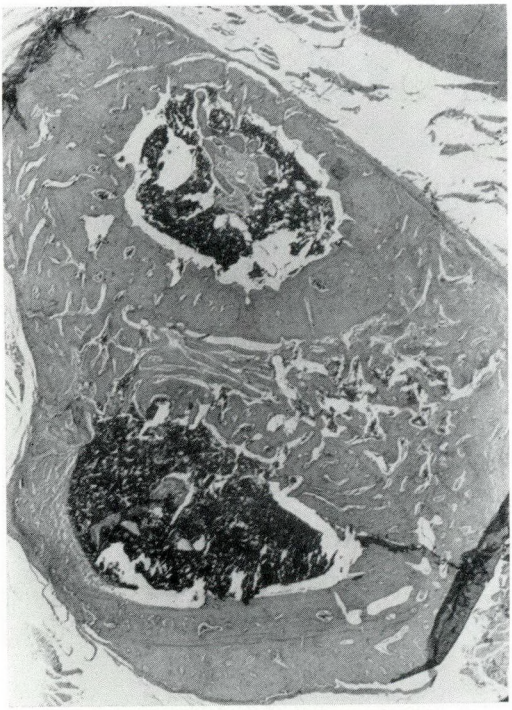

Fig. 11.—Winstrol group five weeks after fracture showing callus completely filled with cancellous bone and bony union. (H & E x 4)

Fig. 12.—Winstrol-cortisone group five weeks after fracture showing complete filling of callus with the cancellous bone and bony union. (H & E x 4)

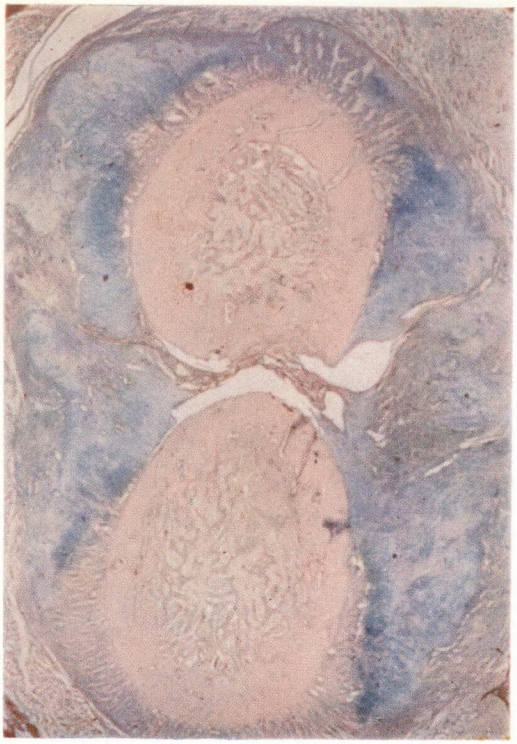

Fig. 13.—Control group two weeks after fracture. The intensity of blue colouration is proportional to the concentration of acid mucopolysaccharides. (Alcian-blue stain x 4)

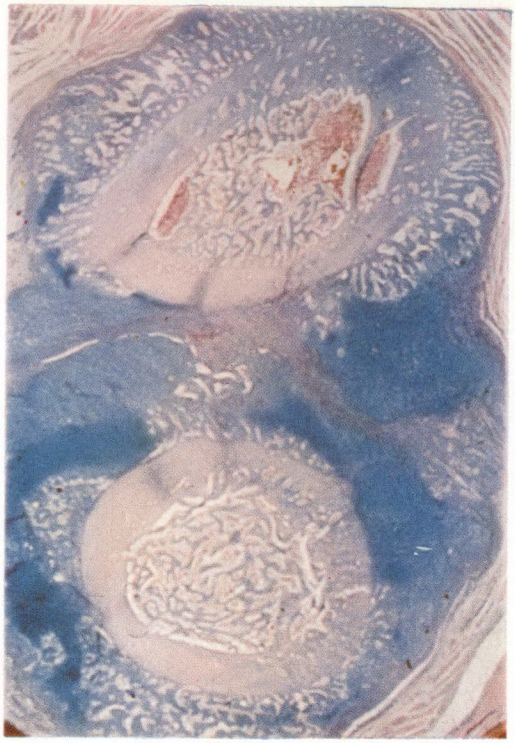

Fig. 14.—Winstrol group two weeks after fracture showing overall increase in blue staining of acid mucopolysaccharides, as compared with control group (Fig. 13). (Alcian-blue stain x 4)

in colour, illustrate the most striking differences in the alcian-blue reaction between the two groups.

Conclusions

In view of the ever-increasing use of corticoids in a variety of clinical conditions, we need more information on the "anti-anabolic" (catabolic) action of the various steroids on bone metabolism and repair. We now have experimental and clinical evidence that, generally speaking, these steroids inhibit connective tissue formation, growth and regeneration. Anabolic steroids have quite the opposite action. They promote connective tissue metabolism and may counteract the inhibitory effects of corticoids under certain conditions.[2]

From the point of view of a clinician interested in bone healing, a delaying or promoting action of a hormone on the normal process of healing appears very important.

Is there any experimental evidence that final bony union of a fracture may be affected by hormones? The present study attempted to answer this question by prolonged observation of the healing process, and by recording progressive clinical, radiological and histological changes at the fracture site. Clinical and radiological delay of healing was found in the cortisone-treated group but the effect of this hormone did not last and final bone union was similar to the one in control animals.

The promoting and/or protective action of the anabolic steroid (Winstrol) was clinically and radiologically observed only in the first weeks of healing. Histologically, inhibition of healing in the cortisone group affected the cellular elements of callus, fibrogenesis, ground substance formation and ossification. The promoting action of Winstrol was opposite to the effects of cortisone, and the protection that this anabolizer provided in groups receiving both steroids was quite remarkable. The effects of steroid treatment were evident in the early stages of healing and did not appear to alter significantly the final issue, bony union.

These results apply to apparently healthy guinea pigs, receiving pharmacological doses of steroids. However, they may suggest to the clinician that the natural healing process of fractured bones should always be considered as a function of time. If the final stage of bony union is, for the clinician, the only important factor, this study does not encourage him to consider steroids as inhibitors or promoters of fracture healing. If however, the clinician is interested in the evolution of the process of healing, which may be altered in such conditions as shock, malnutrition, senile osteoporosis, prolonged cortisone therapy, etc., the results of this study may be valuable. Particularly interesting is the observation that clinically, radiologically and histologically, an anabolizer does protect a healing fractured bone against the inhibitory action of cortisone during the first three weeks after fracture, under the experimental conditions described.

Summary

Guinea pigs were treated with anti-anabolic cortisone and an anabolic androgen before fracture of the humerus and during the period of healing. The effects of these steroids on the clinical, radiological and histological process of healing has been studied. Observations were recorded on the fourth day and at the end of the first, second, third, fourth, fifth and sixth week after fracture. The inhibitory effects of cortisone on clinical, radiological and histological healing were observed during the first two weeks and these effects were counteracted when both steroids were given simultaneously. The anabolic steroid, Winstrol, used in this study promoted early stages of histological healing and protected against the effects of cortisone.

These steroids affected the cellular elements of callus, fibrogenesis, cartilage formation and ossification, and were only apparent for the first two to three weeks of the healing period.

No significant differences between the experimental groups were detected in healing humeri, by methods used in this study, at the end of the fourth, fifth and sixth week after fracture. The possible clinical significance of these observations is discussed.

The authors would like to express their appreciation to Winthrop Laboratories of Canada, Aurora, Ont. for a research grant and supply of the anabolic steroid which made this work possible. Merck Company of Canada is thanked for the supply of Cortone. The technical assistance of Mrs. A. Aksel, D.V.M. and Mr. E. Burton is acknowledged.

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RÉSUMÉ

On a traité des cobayes au moyen de cortisone anti-anabolique et d'un androgène anabolique avant fracture de l'humérus et pendant la période de guérison. On a noté les effets de ces stéroïdes sur l'évolution clinique, radiologique et histologique de la consolidation, au quatrième jour et à la fin des première, deuxième, troisième, quatrième, cinquième et sixième semaines après la fracture. Les effets inhibiteurs de la cortisone sur la guérison clinique, radiologique et histologique ont été notés durant les deux premières semaines de la période d'observation. Le stéroïde anabolique, (Winstrol), a favorisé la guérison histologique, durant les premiers stades, et a permis de protéger le malade contre les effets de la cortisone.

Les effets de ces stéroïdes concernaient les éléments cellulaires du cal, la fibrogénèse, la formation du cartilage et l'ossification.

On n'a pu déceler de différences notables entre les groupes expérimentaux, au point de vue de la consolidation de l'humérus, du moins par les méthodes employées dans la présente étude, à la fin des cinquième et sixième semaines après la fracture.

LANGUAGE AND THE PHYSICIAN

Students of science often have a cavalier attitude toward instruction in use of the language, which they patronizingly and mistakenly label "English grammar". They seem to consider it beneath their professional dignity to study linguistics and composition. After all, they are going to be specialists. What do they need to know about the language that they have not already absorbed? They know the lingo, the cant, the jargon that marks them as an insider. But the average medical graduate fails to realize that for four years he has lived primarily among his own professional species and has had the sympathetic eyes and ears of his teachers, who already understand the ideas he wishes to convey. Once he emerges from the cloistered walls of the medical school and hospital, however, he will have to speak in the native tongue of those who do not understand medicine—in plain English. And he cannot risk being misunderstood by them.

As the physician matures, moreover, he will realize that it is important *how* he says things, and that clear, concise, forceful English is *masculine* by its very strength. When he is first obliged to write a report for admission to a professional organization or for delivery before his colleagues, he will suddenly wish that he had not considered self-expression a negligible accomplishment. He will then have to tackle alone that long-neglected aspect of his education—the effective communication of ideas in speech and writing.

For four years the medical student's reading is limited, perhaps almost exclusively, to medical publications (erroneously termed *literature*), which are marred by vagueness, repetition, monotony, ambiguity, confusion, and actual misstatement. We can hardly expect the apprentice medical writer to strive for literary excellence when the standards to which he has been exposed for four years have been far from adequate. The medical student becomes so accustomed to medicalese that he recognizes no flaws in it; he may even consider it the exclusive property of the initiated that need communicate to no one else. Rarely does he examine critically words that slip glibly from his tongue or his pen. He uses peculiar expressions without any thought of what they really mean. His statements may be so absurd that the listener must supply the intended meaning himself, as in the statements:

No tumour is hopeless unless proved otherwise.

His pelvis was fractured by being thrown from an automobile.

While passing a stomach tube, the trachea was inadvertently entered.

Often the reader or listener is left to *guess* what the intended meaning is. This becomes a crucial matter when the patient needs an interpreter to undertand his physician. Despite contentions to the contrary, cogent thinkers do not always write and speak clearly, although lucid language can emanate only from an orderly, clear mind.—L. DeBakey: Language and the Physician, *Arch. Surg. (Chicago)* **92:** 964, 1966.

THE RESULTS OF USING METHYL 2-CYANOACRYLATE MONOMER IN EXPERIMENTAL SURGERY*

S. E. CARROLL, M.D., F.R.C.S.[C],† A. L. AMACHER, M.D.‡ and J. C. PATERSON, O.B.E., M.D.,§ *London, Ont.*

MAINTAINING patency of small-vessel anastomoses, particularly in veins, remains a surgical problem.[1, 2] Previous studies in this laboratory have shown a high success rate in femoral vein grafts in dogs using the NRC-Vogelfanger stapling device.[3] In the light of many reports[2, 4-6] concerning the use of tissue adhesives, the present study was undertaken to assess the value of methyl 2-cyanoacrylate monomer¶ as an adjunct to improve the results of small-vessel anastomoses by improved hemostasis, especially in heparinized animals. A subsequent increase in patency rates should be obtained. The results of this three-phase investigation proved to be disappointing.

TABLE I.—NECROSIS IN FEMORAL VESSELS IN CATS

	ARTERY			*VEIN*		
	No. of vessels	*Necrosis present*	*Severe necrosis present*	*No. of vessels*	*Necrosis present*	*Severe necrosis present*
Monomer	10	9	6	10	4	2
Suture	10	4	1	10	6	1
Monomer and suture	9	7	5	9	6	4
Suture and heparin	10	8	2	10	6	2
Total	39			39		

PROPERTIES OF THE MONOMER

Methyl 2-cyanoacrylate monomer[7] is a clear liquid that polymerizes within seconds upon exposure to moisture, metals, pressure or heat. It must be stored in sealed plastic containers, not allowed to touch the skin and causes instruments to become glued together. A solvent is available but is toxic. Unlike the parent substance, Eastman 910 adhesive, the monomer contains no thickening agent, plasticizer or inhibitor, and its viscosity is low. The additives are not recommended for human use since they may be carcinogenic. The monomer is self-sterilizing but deteriorates upon exposure to air. Individual vials of the monomer used in these experiments were kept as long as two weeks in a refrigerator.

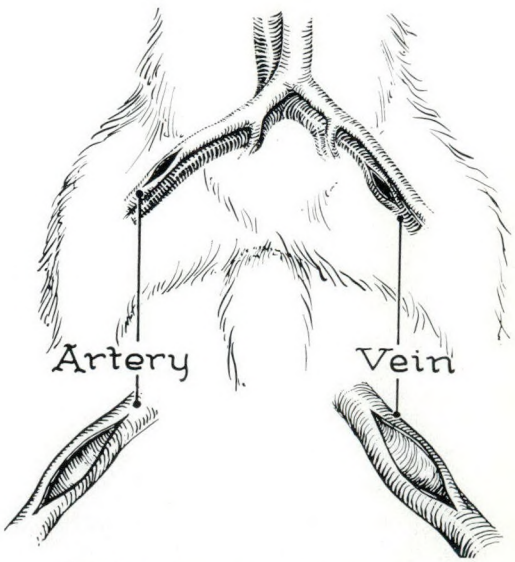

Fig. 1.—Preliminary studies in cats.

*From the University of Western Ontario Medical School and Westminster Hospital (DVA), London, Ont.

†Part-time Research Associate of the Ontario Heart Foundation, Consultant Surgeon, Westminster Hospital (DVA), London, Ont.

‡Fellow in Surgery, University of Western Ontario, London, Ont.

§Chief of Laboratory Services, Westminster Hospital (DVA), London, Ont.

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Supported by the Ontario Heart Foundation.

¶Generously supplied by Ethicon Inc., as Eastman 910 monomer.

TABLE II.—Tissue Reaction to Injected Monomer in Cats

CONTROL (saline)				*MONOMER*		
Cat number	*Site*	*Degree of necrosis*	*Tissue reaction*	*Site*	*Degree of necrosis**	*Tissue reaction*
1	Right thigh	0	0	Left thigh	+	Chronic inflammation, slight edema
	Right abdominal wall	0	0	Left abdominal wall	+++	Acute inflammation
2	Left thigh	0	0	Right abdominal wall	+++	Acute inflammation
				Right thigh	+++	Acute inflammation
3	Right thigh	0	0	Left abdominal wall	+++	Moderate acute inflammation
				Left thigh	+++	Acute and chronic inflammation
4	Left thigh	0	0	Right abdominal wall	+++	Acute peritonitis, inflammation of muscle coats
				Right thigh	+++	Severe acute inflammation
15	Abdominal wall, thigh	0	0	Abdominal wall	+++	Acute inflammation
				Thigh	+	Slight degeneration
19	Left thigh	0	0	Right thigh	0	Deposit not found
20	Thigh	0	0	Right abdominal wall	+	Ulcer, severe edema of muscle, slight inflammation
				Thigh	+++	Acute inflammation
21	Thigh	0	0	Right thigh	++	Acute inflammation of muscle
				Abdominal wall	+++	Peritoneum entered. Peritonitis, perienteritis

* + = slight, ++ = moderate, +++ = severe necrosis.

Preliminary Studies in Vascular Closure

Using anesthetized cats, under clean surgical conditions, incisions 1 cm. long were made in one femoral artery and the opposite femoral vein (Fig. 1). These incisions were closed with monomer alone, vascular suture alone or sutures combined with monomer. In a fourth group, the incisions were sutured and the cats heparinized with a single dose of Depo-heparin (heparin sodium). The type of closure was randomly selected. After seven days, angiography was carried out by injecting contrast media into the external iliac artery and saphenous vein. The animals were then sacrificed.

The results, in terms of histological evidence of necrosis in the vessel walls, are shown in Table I. The degree of necrosis in each vessel was graded from + (slight) to +++ (severe) by an independent observer who was not aware of the type of closure.

This part of the study demonstrated that:

(1) There were no significant differences in the percentage of vessels remaining patent in these four groups.

(2) Five of 10 femoral arteries closed with monomer alone developed arterial hematomas.

(3) There was no difference in the incidence of wound infections or mortality in the four groups.

(4) Use of the monomer did not decrease the need for additional sutures in the incisions to obtain hemostasis.

(5) Necrosis of the vessel wall, particularly of severe degree, was more common when the monomer was used, although the differences were not significant.

The high rate of necrosis and marked inflammatory response with Eastman 910

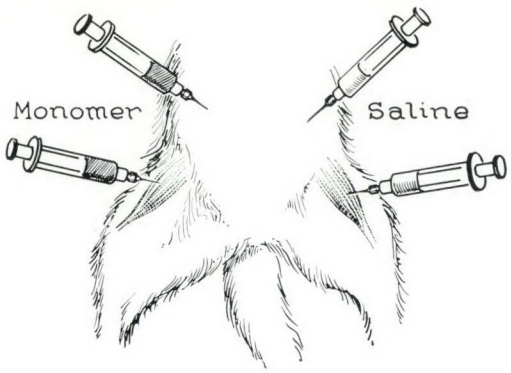

Fig. 2.—Injections in cats.

monomer in vascular closure suggested the need for reassessment of the effects of the monomer upon other tissues.

Studies of Tissue Reaction to Eastman 910 Monomer

Under ideal conditions, foreign material implanted in tissue elicits a minimal inflammatory reaction, provided infection was absent. Necrosis in the host tissue should be absent or slight.

Injection into Tissues

In eight prepared cats, 0.5 to 2 c.c. of monomer was injected into the abdominal wall and thigh muscle of one side, using plastic disposable syringes (Fig. 2). Similar injections of sterile normal saline were made on the opposite side of each animal. The animals were sacrificed after seven days and sections prepared from the injection sites. The results of histological assessment of the effects of injected monomer is shown in Table II. Many of the sites of saline injection could not be located grossly or microscopically, hence the deficiencies in the control series of Table II. The deposits of monomer were stony hard, and adherent to surrounding tissue. It is plainly seen (Fig. 3) that the monomer produced necrosis of muscle and other tissue. One cat, in which the peritoneum had been punctured, developed peritonitis (Fig. 4) and inflammation of the muscle coats of the small bowel. Another cat died of chemical peritonitis four days after injection.

External Application To Veins

To assess the effects of the monomer upon vessel walls, both femoral veins were exposed but otherwise undisturbed in 10 anesthetized cats. A single drop of monomer was applied externally to one femoral vein, a drop of saline to the opposite vein. The wounds were closed in layers. After seven days the animals were sacrificed and both femoral veins removed for histological assessment.

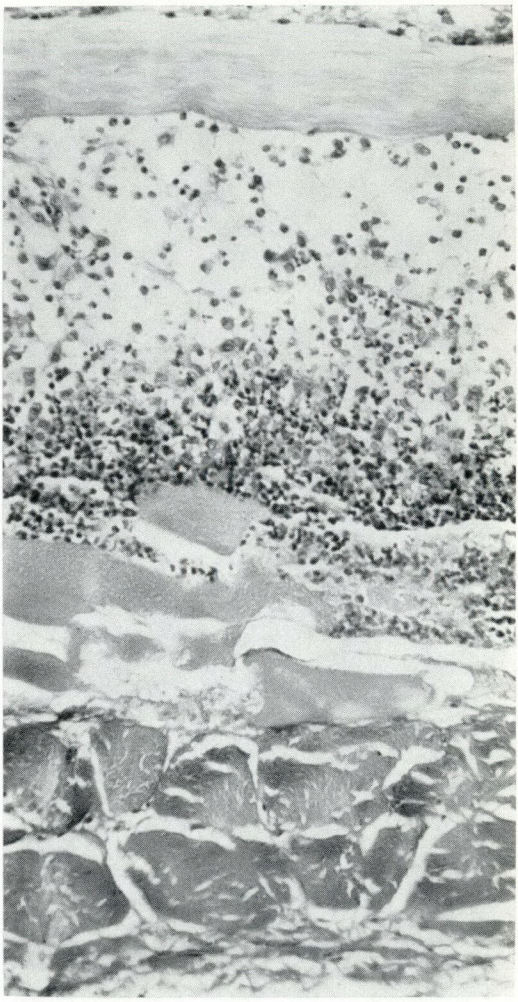

Fig. 3.—Monomer injection site in abdominal wall. Note necrosis of muscle and severe inflammatory reaction.

The results of this experiment are shown in Table III. Healing by first intention occurred in all 10 wounds treated with saline. Six of the 10 wounds receiving the monomer were open, with a cavity in the wound (one example is shown in Fig. 5a). All 10 of the latter exhibited induration and gross inflammation.

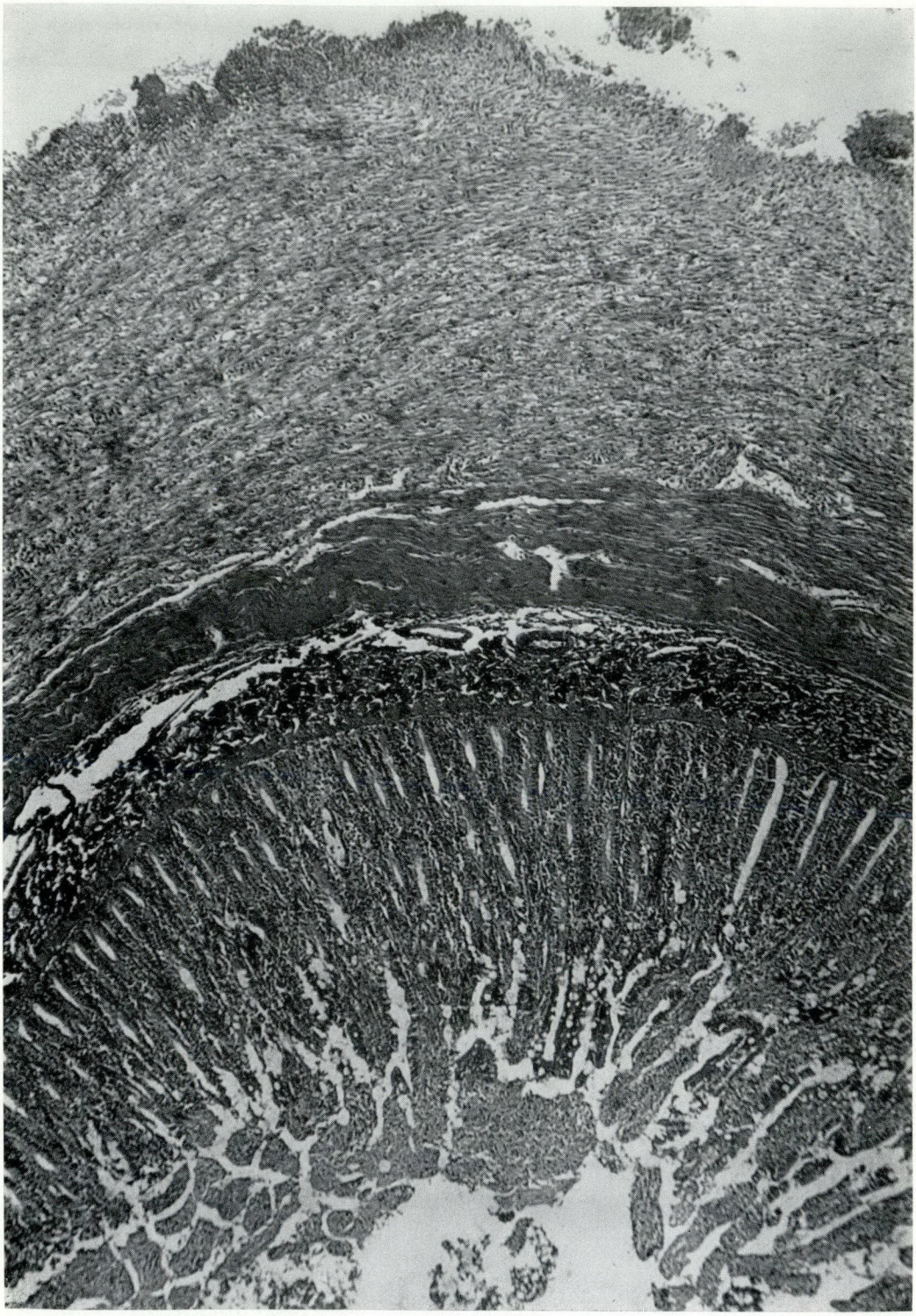

Fig. 4.—Chemical peritonitis produced by monomer. Note severe peritoneal reaction and inflammation in the muscle coats.

TABLE III.—Reaction of Veins to External Application of Monomer

Cat number	CONTROL (saline)		MONOMER	
	Gross	Microscopic	Gross	Microscopic
65	Wound healed. Little reaction	Normal vein	Ragged sloughing wound. Vein intact	Normal vein
66	Wound healed. Very little reaction	Focal acute periphlebitis. Slight diffuse inflammation of media with proliferation	Wound open. Tissue reaction heavy	Acute periphlebitis. Focal acute inflammation of media
67	Wound healed	Normal vein	Wound open. Marked skin reaction	Slight periphlebitis only
68	Wound healed. Little tissue reaction skin	Normal vein Normal valve	Wound open. Marked tissue reaction. Necrotic cavity around vein	Severe necrosis and acute inflammation. Vein not identified
69	Little reaction in wound	Normal vein	Marked tissue reaction. Cavity around vein	Necrosis and acute inflammation of half circumference, very severe
70	Wound healed	Normal vein	Wound open. Marked tissue reaction	Severe necrosis. Inflammation of vein wall. Moderate periphlebitis
71	Wound healed	Normal vein	Wound open. Skin open. Much tissue reaction	Severe periphlebitis. Slight acute inflammation with early necrosis
72	Wound healed	Normal vein. Dropping out of muscle nuclei	Marked tissue reaction	Focal periphlebitis. Focal acute inflammation of vein wall in two spots. Severe necrosis
73	Little reaction	Normal vein	Marked reaction	Normal vein
74	Wound healed	Normal vein	Marked reaction	Vein shows segmented necrosis. Acute inflammation
Totals	10 Primary healing 10 Slight reaction	9 Normal veins 1 slight change	6 Open wounds 10 Marked reaction	2 Normal veins 6 Necrotic veins 8 Acute inflammation

Histologically, nine of the 10 veins treated with saline were normal and one demonstrated slight changes. Conversely, of 10 veins to which the monomer had been applied, only two were considered normal; six had necrosis of the vein wall and eight demonstrated acute inflammatory changes in the tissues surrounding the vein (Fig. 6).

Studies in Canine Vein Grafts

To exclude the factor of species variation and to allow comparison with previous experimental results obtained in a study of canine vein grafts, the monomer was tested in the following manner. Simultaneous bilateral vein grafts were performed in anesthetized mongrel dogs under surgically clean conditions (Fig. 7). A 4-in. segment of one external jugular vein was removed, irrigated with heparinized saline and divided. One portion was transplanted to the occluded, divided superficial femoral vein, using the NRC-Vogelfanger stapling device as previously described.[1, 3] The second portion was transplanted to the opposite superficial femoral vein in similar fashion. On one side, selected randomly, the cuffs were everted after stapling and one

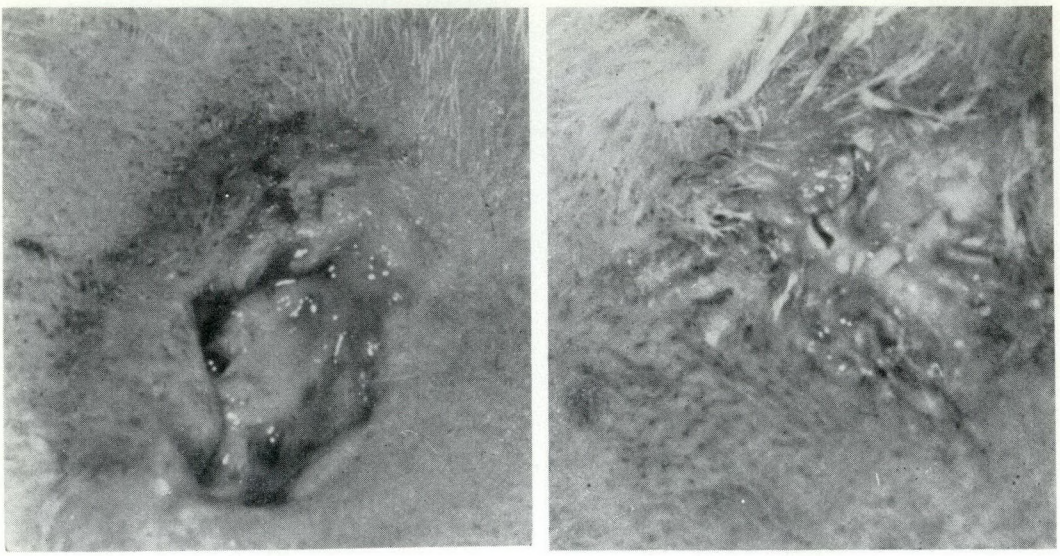

Fig. 5a **Fig. 5b**

Fig. 5.—Wound reaction to monomer applied externally to femoral veins. (a) Note the cavity produced in the wound where one drop of monomer has been used. (b) The wound has healed in the same cat at seven days with saline applied.

TABLE IV.—Reaction to Monomer of Bilateral Vein Grafts in Dogs

	CONTROL		*MONOMER*	
Dog number	*Necrosis in graft wall*	*Perivascular reaction*	*Necrosis in graft wall*	*Perivascular reaction*
567	0	+++	0	+++
566	0	++	+	—
563	0	+++	0	+++
575	0	++	0	+
574	0	+	0	++
614	+++	++	Not identifiable (+++)	+++
618	+++	—	+++	++
379	0	—	++	—
377	No section	—	+	+++
378	0	++	0	++
372	0	+++	++	++
380	0	+	0	+
362	+	++	+	+++
357	0	0	0	0
339	0	0	+++	—
338	0	0	0	0
426	0	0	+	—
404	+++	+	+	—
384	+	—	+	—
421	0	++	0	++
408	0	—	0	—
447	+	—	+++	—
401	0	—	+++	—
617	++	—	Not identifiable (+++)	+++
619	+	—	+	—
667	No section	—	+++	—
356	0	—	0	—
Total	8/25	13/27	16/27	13/15

+ = slight, ++ = moderate, +++ = severe necrosis.

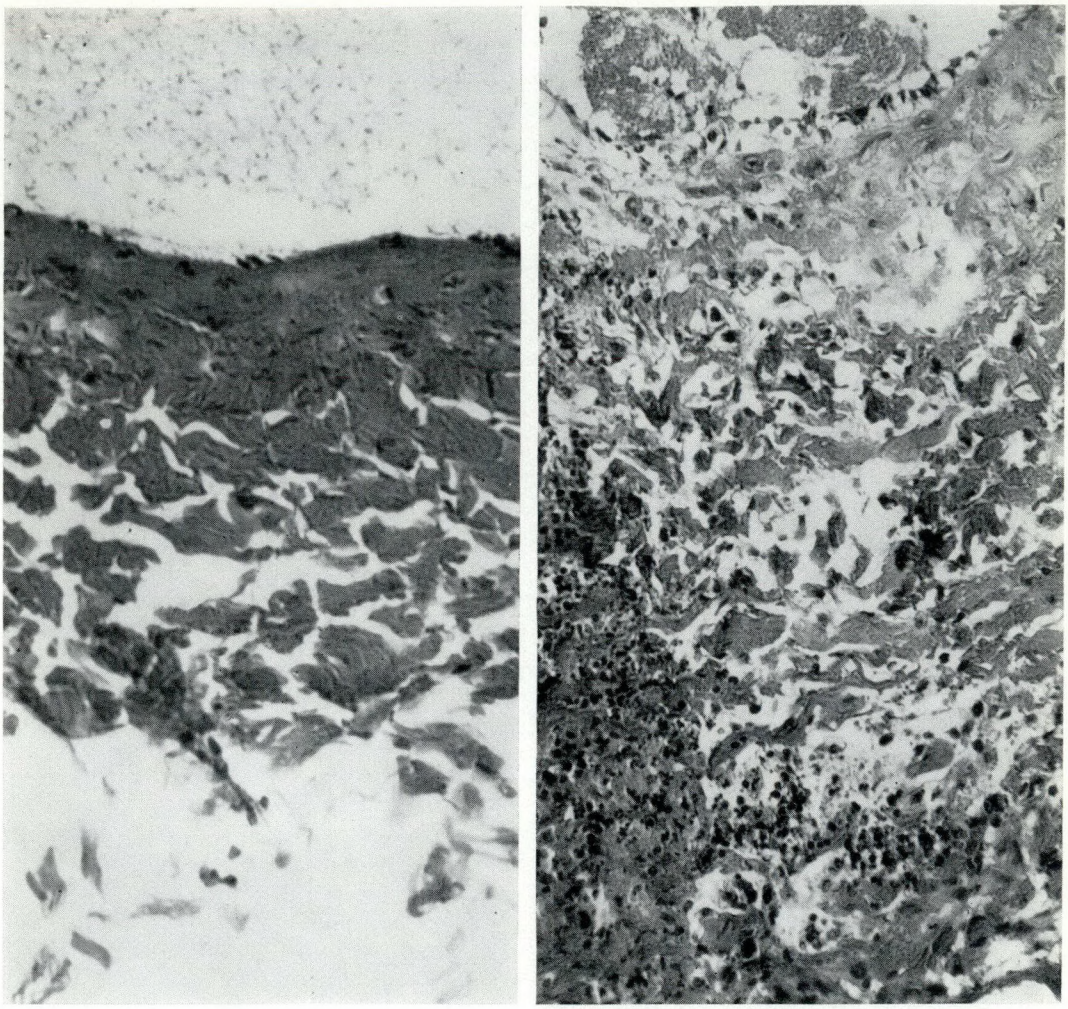

Fig. 6a Fig. 6b

Fig. 6.—Vein reaction to external application. (a) Normal feline vein, saline applied seven days previously. (b) Necrosis of vein wall with severe inflammatory reaction. Monomer applied seven days before sacrifice.

drop of monomer was applied with a thinned artists' brush (Fig. 8) to the intimal surfaces.

The anastomoses on the monomer-treated side were accomplished first in 18 of 28 dogs, since the duration of graft ischemia is considered an important factor in successful grafting. All wounds were closed in layers and the animals heparinized for 36 hr. After seven days, femoral venograms (Fig. 9) were obtained and the animals sacrificed. Histological sections of the grafts were taken to compare the degree of tissue reaction on the two sides (Fig. 10).

The patency rate in the grafts treated with monomer was 32% and in those treated with saline was 57%. While these results are worse than a previous group of 12 autogenous femoral grafts in which all were patent at seven days in heparinized dogs,[3] the prolonged time of graft ischemia is considered a sufficient explanation for the difference.

Grossly, all wounds healed without infection. In several dogs a cavity was found surrounding the anastomoses in which monomer had been used. Such a cavity would compromise vein grafts in humans.

The tissue reactions in the vein grafts and in the surrounding tissues are summarized

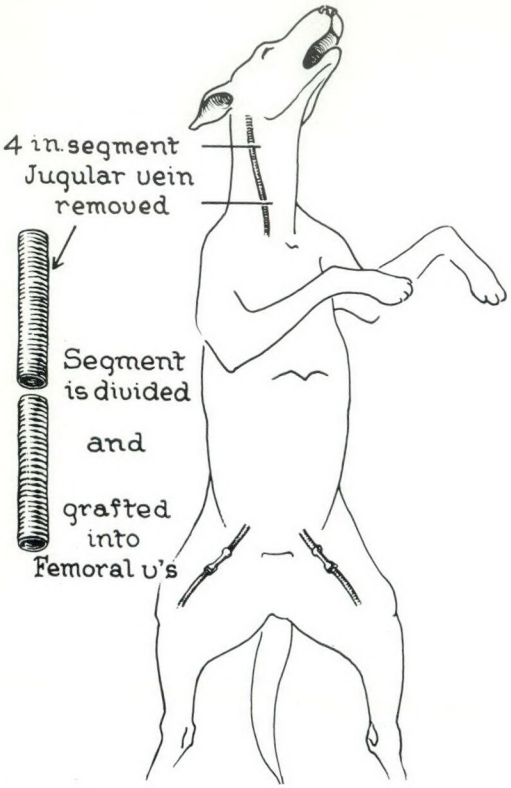

Fig. 7.—Simultaneous bilateral femoral vein grafts.

in Table IV. Necrosis of the graft was observed in eight of 24 saline controls and in 16 of 27 grafts receiving the monomer. This difference is not statistically significant. There was little difference in the amount of perivascular reaction.

Discussion

A tissue adhesive that was safe from the standpoint of infection and carcinogenesis, that acted rapidly to save operating time, and was strong and durable enough to withstand distension by arterial blood pressure, would be very desirable. In vascular surgery, such an ideal substance might eliminate sutures entirely or decrease the number of stitches required, thus shortening the duration of operation and particularly the duration of interruption of blood flow. The amount of blood lost would be less and greater success might be obtained in such difficult problems as anastomosis of small veins. Methyl 2-cyanoacrylate monomer has been proposed for these purposes. Small veins were used to test the monomer because they are thin-walled and susceptible to injury.

The difficulties in handling this material, which polymerizes rapidly and glues instruments or digits together, are well known. The low viscosity of the monomer made it impossible to use it in smaller quantities on these small vessels.

In these experiments, the monomer was tested in cats and dogs. Necrosis of veins, muscle, bowel and connective tissue was produced by the monomer and a marked inflammatory response elicited. It is reasonable to assume a similar response would occur in humans and this would likely lead to occlusion of vein grafts.

It is our opinion that a substance that produces necrosis should not be recommended for use in human beings, although small quantities in a large diseased artery deep in the body might be tolerated.

Although early reports[2, 5, 7] on experimental use of this material were promising, several recent publications[8, 9] describe difficulties encountered with it and similar materials.

In transplanting vein grafts in dogs, bleeding from the anastomoses was anticipated in heparinized animals. It seemed reasonable to use a tissue adhesive to close gaps between staples if necessary, rather than sutures since the intimal surfaces were everted and there would be no contact with the blood stream. In fact, hemostasis did not present a major problem in these experiments. When bleeding was encountered, the use of the monomer was awkward and was less effective than placing an additional suture in the anastomotic line.

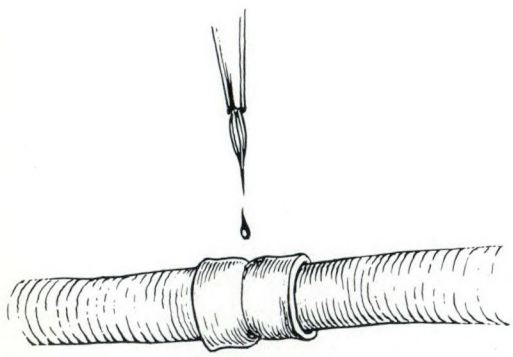

Fig. 8.—Application of monomer.

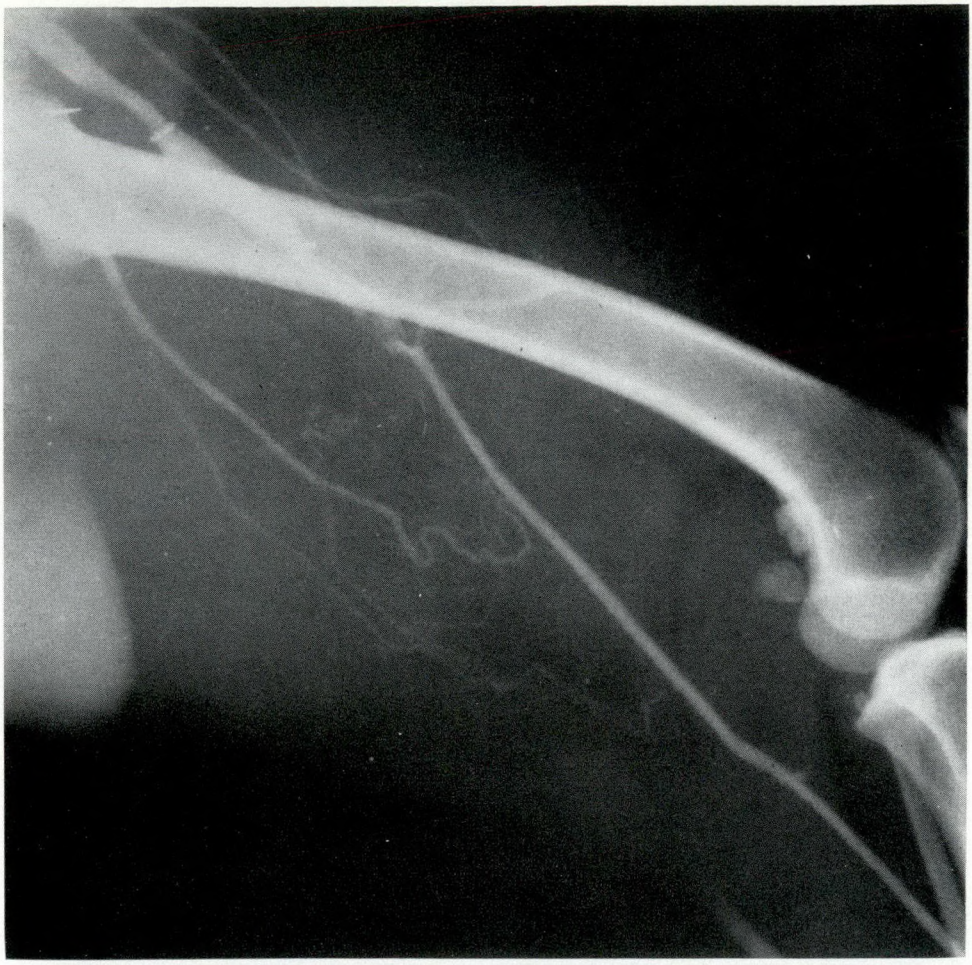

Fig. 9.—Representative femoral venogram. Hypaque injected in the saphenous vein has entered the femoral and iliac veins. Note the patent graft between ring of staples.

Conclusions

In these experiments the application of methyl 2-cyanoacrylate monomer to incised vessels and anastomotic sites did not improve hemostasis or the patency rate. Eastman 910 monomer produced marked tissue reaction in various tissues of the cats.

The monomer produced necrosis in the wall of the femoral veins in cats and in vein grafts inserted into dogs' femoral veins.

Methyl 2-cyanoacrylate monomer is not recommended for use in human surgery.

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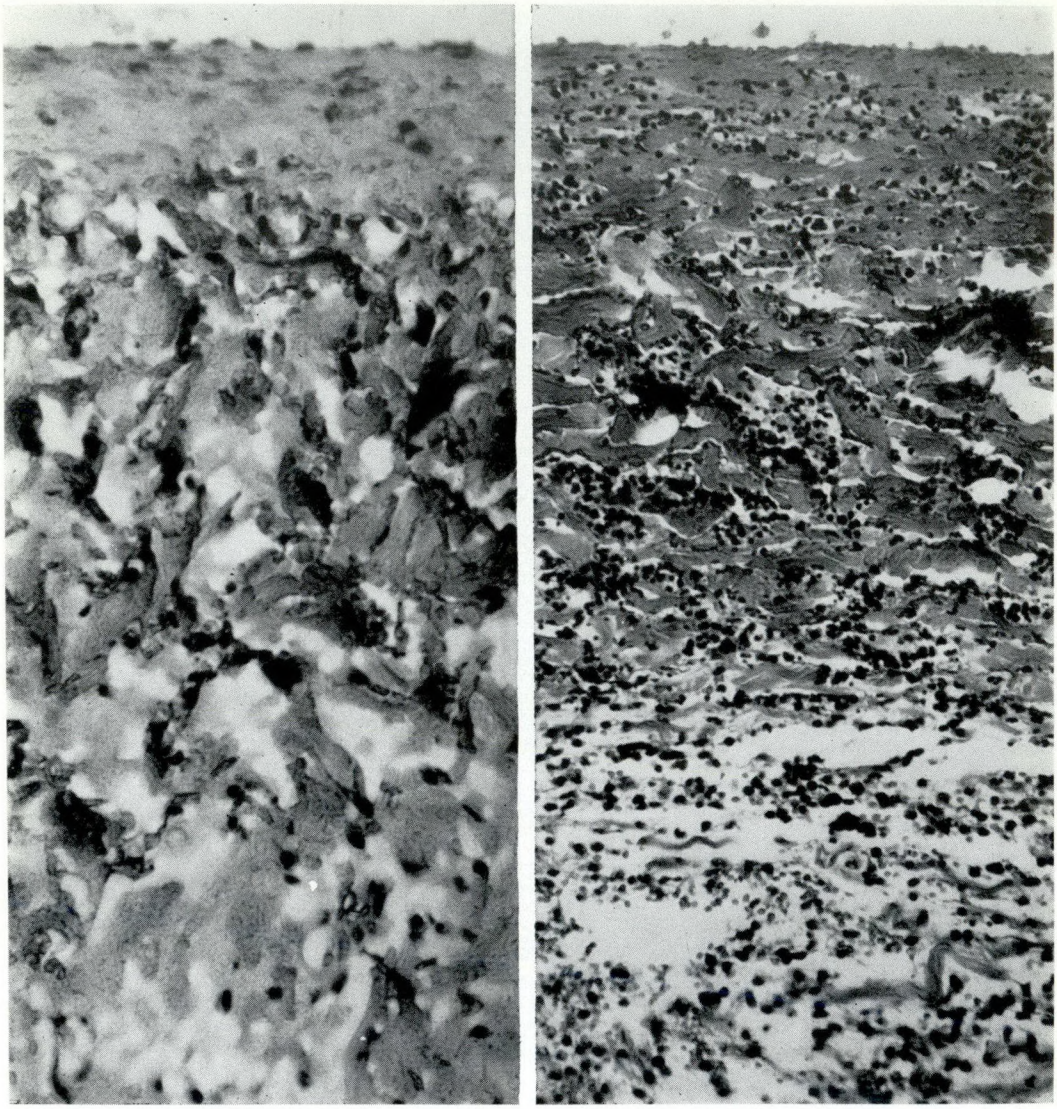


Fig. 10a **Fig 10b**

Fig. 10.—Reaction of vein grafts to monomer. (a) A section of canine vein graft, seven days after application of saline. There is slight (+) intimal thickening, slight (+) inflammation of the wall and slight (+) necrosis. (b) A similar section after monomer application. There is marked (+++) inflammation of the wall and severe (+++) necrosis.

Résumé

Un adhésif neutre pour la fermeture des vaisseaux constituerait un nouvel instrument très précieux dans l'arsenal de la chirurgie vasculaire. Etant donné les nombreux rapports consacrés à l'emploi de ce genre d'adhésifs en chirurgie expérimentale, nous avons essayé le méthyl 2-cyanoacrylate monomère chez des chats et des chiens au cours d'expérience comportant des témoins; (a) en application sur des vaisseaux incisés; (b) en injection dans les tissus, et (c) pour consolider des anastomoses dans des greffes veineuses.

Une polymérisation du monomère a fait naître des difficultés techniques. Son application à des artères et des veines de chats et de chiens a provoqué de la nécrose et des hématomes dans les artères. Le monomère, injecté à un second groupe de chats a entraîné de la nécrose tissulaire et de l'inflammation aiguë, à l'inverse des injections de sérum physiologique chez les sujets-témoins. Des modifications semblables ont survenu par application du monomère à des veines intactes chez le chat. L'emploi du monomère sur les lèvres des anastomoses suturées au niveau des greffes des veines fémorales chez le chien n'a pas amélioré le degré de l'ouverture ni l'hémostase. On déconseille d'employer le méthyl 2-cyanoacrylate monomère en chirurgie humaine.

EDITORIAL

BLUNT ABDOMINAL TRAUMA

Blunt abdominal trauma, a perplexing surgical problem, is difficult to manage well because it is difficult to recognize early. In many instances, a history of significant abdominal injury is lacking and, even when such a history is obtained, it may be clinically difficult to establish that trauma, justifying exploration, has occurred. Until recently less than 0.1% of general hospital admissions were due to blunt abdominal trauma but, with the inexorable increase in traffic accidents, this percentage will increase. On Canadian highways, in 1964, 139,534 people were injured and 4655 killed: an 11.5% increase in injuries and a 10.6% increase in deaths over the previous year. From the 1964 experience it has been estimated that, in that year, some 3000 Canadians received abdominal injuries in motor vehicle accidents and, in 1500, such injury was the sole or contributing cause of death.

Canadian experience with blunt abdominal trauma in Edmonton and Ottawa is summarized elsewhere in this issue. Macbeth (p. 384) reviewed the records at the University of Alberta Hospital in Edmonton over a 14-year period and found 100 patients with such injuries; 62% were due to motor vehicle accidents. In the same period in Edmonton, only 13 patients were seen with penetrating abdominal injury and, of these, the injuries of all but two were accidentally inflicted. This is in marked contrast to the experience in United States cities where penetrating abdominal injuries predominated. In Texas, for example, 90% of hepatic injuries were due to penetrating trauma.

The criteria for blunt abdominal trauma in Macbeth's study were as follows: (1) the patients lived to be admitted to hospital; (2) the diagnosis was confirmed at operation or autopsy; and (3) traumatic lesions to the urinary tract were excluded. The injuries in these 100 patients were as follows: splenic, 53; hepatic, 17; combined splenic and hepatic, nine; gastrointestinal perforation, 10; pancreatic, five; mesenteric laceration, four; and retroperitoneal hematoma, two. The overall mortality rate was 17% and varied from 89% (combined splenic and hepatic injury) to 7.5% (splenic rupture).

McLeod and Brown (p. 379) at the Ottawa Civic Hospital reviewed 110 patients treated for proved intra-abdominal injuries over a five-year period, January 1960 to December 1964. In this series there were 86 males and 24 females, a ratio of almost four to one. There were 11 deaths, a mortality of 10%. The highest incidence of injury was in the age group, 10 to 30 years, but the mortality rate was significantly higher at the extremes of life. Once again, the greatest number of injuries and the highest mortality was due to motor vehicle accidents.

In the Ottawa series, trauma to other organ systems was frequently associated with intra-abdominal injury; 61 patients (55%) had associated injuries. Twenty-six patients had thoracic injury and 24 had significant head injuries. As would be expected, the highest mortality rate was seen in patients with combined head, thoracic and abdominal injuries. Where only intra-abdominal organs were involved, there were no deaths; where extra-abdominal injuries were present, the overall mortality rate was 16%; but when head, thorax and abdomen were injured, the death rate was 57%.

The mortality rate in penetrating wounds of the abdomen is 7 to 10% while the rate due to blunt abdominal trauma remains at 10 to 30%. There are good reasons for this discrepancy. In civilian practice a penetrating wound of the abdomen is almost always an isolated injury. Because it is obvious to the patient that the wound may have serious implications, medical help is usually sought immediately; exploration of the abdomen is mandatory and is carried out as soon as the condition of the patient permits. Aside from the fact that the other

injuries, which are so commonly associated with blunt abdominal trauma, may themselves lead to death or be a major contributing factor, there remains the difficulty of recognizing the non-penetrating abdominal injury early. As McLeod and Brown pointed out, "Unlike penetrating wounds, injuries caused by blunt trauma, even when severe, are frequently not obvious. Physical findings may be lacking, obvious injuries to other parts of the body may obscure the abdominal trauma and, if the associated injury requires an emergency operation under general anesthesia, the signs and symptoms of intra-abdominal injury may be further suppressed. Under these circumstances an early diagnosis may be virtually impossible . . ."

The kind of accident that may cause abdominal trauma can happen anywhere, and will do so with increasing frequency. All physicians and surgeons who take responsibility for the emergency care of injured patients must remain alert to recognize these injuries, to carry out adequate resuscitation and early, appropriate treatment. Macbeth emphasizes that an accurate history of how the accident happened may not only suggest the probability of an intra-abdominal injury but may even indicate the quadrant of the abdomen affected. When the evidence suggests a reasonable probability of intra-abdominal injury, the surgeon must accept the responsibility for exploration and not feel apologetic when, on occasion, this proves to be negative.

The Royal College of Physicians and Surgeons of Canada

NEWSLETTER

1. Regional Meetings

Western Regional Meeting

A Western Regional Meeting of The Royal College of Physicians and Surgeons of Canada will be held at the Bessborough Hotel in Saskatoon on November 24 and 25, 1966. The meeting is designed primarily for Fellows and Certificants of the College in Area II—Saskatchewan and Manitoba, though Fellows and Certificants from all areas will be welcomed. Some of the features will be half-day symposia on shock, and cardiac arrhythmias, with outstanding guest participants. The program will also include a number of scientific papers by Fellows and Certificants from Area II. Enquiries concerning the meeting should be directed to the Chairman of Local Arrangements, Dr. Allan A. Bailey, Department of Medicine, University Hospital, Saskatoon.

Ontario Regional Meeting

A Regional Meeting of The Royal College of Physicians and Surgeons of Canada will be held at Laurentian University in Sudbury, Ontario, on November 11 and 12, 1966. A program of high calibre with a number of distinguished guest speakers is being arranged. The meeting is primarily designed for Fellows and Certificants of the College in Area III—Ontario, but Fellows and Certificants from all areas in Canada are free to attend and will be welcomed. Application for hotel accommodation should be made to the President Hotel, Elm Street, Sudbury, Ontario. Further enquiries concerning this meeting should be directed to the Chairman of Local Arrangements, Dr. Bruce M. Wilson, 137 Cedar Street, Sudbury, Ontario.

2. 1967 Annual Meeting

It is appropriate that the Annual Meeting of the College in Canada's centennial year should be held in the nation's capital. The Meeting will take place at the Chateau Laurier, January 19, 20 and 21. Plans for the scientific program are well advanced and another outstanding meeting is prom-

ised. While facilities, particularly hotel accommodation, is not as abundant or as convenient as in the larger centres of Montreal and Toronto, it is hoped this will not be a deterrent to attendance. Good hotel accommodation can be assured to all those wishing to attend. Hotel application forms have already been mailed to the Fellows. If you have not received yours or have misplaced it, an additional copy can be obtained by writing to the College office.

3. Invitation to Certificants of the College

As in recent years, a cordial invitation is extended to all certificated specialists of the College wishing to attend the Annual Meeting. Those desiring to do so are requested to register in advance by completing and forwarding the Registration Form below.

W. Gordon Beattie, F.R.C.S.[C],
Honorary Assistant Secretary

The Secretary,
The Royal College of Physicians and Surgeons of Canada,
74 Stanley Avenue,
Ottawa 2, Ontario.

I desire to register to attend the Scientific Sessions of the Annual Meeting of The Royal College of Physicians and Surgeons of Canada to be held at the Chateau Laurier Hotel, Ottawa, January 19, 20 and 21, 1967.

Enclosed is a cheque/money order in the amount of $15.00 in payment of the Registration Fee.

Name of Certificant .....

Address .....

.....

Name of Specialty .....

(Please print)

INTERNATIONAL SYMPOSIUM ON PHYSICAL ACTIVITY AND CARDIOVASCULAR HEALTH

An International Symposium on Physical Activity and Cardiovascular Health is being held in Toronto, Ontario, on October 11 to 13, 1966.

This symposium, jointly sponsored by the Ontario Heart Foundation and the Ontario Medical Association, will consider (a) current interpretations of fitness, including assessment, determinants and changes during training; (b) fitness and the prevention of cardiovascular disease in animals and man; and (c) planning for cardiovascular health including activity patterns, a consideration of rehabilitation and motivation.

For information contact the Ontario Heart Foundation, 247 Davenport Road, Toronto 5, Ontario.

THE CANADIAN JOURNAL OF SURGERY

All communications concerning this Journal should be marked "The Canadian Journal of Surgery" and addressed to the Editor, C.M.A. Publications, at C.M.A. House, 150 St. George St., Toronto 5.

The Journal is published quarterly. Subscription is $10 per year ($5 per year for trainees in surgery), and starts with the January issue of each year. Single copies are $2.50 each, payable in advance. (It would be greatly appreciated if subscribers would please add bank exchange to their cheques.)

INSTRUCTIONS TO CONTRIBUTORS

Manuscripts

Manuscripts **in duplicate** of original articles, case reports, and other contributions should be forwarded with a covering letter requesting consideration for publication in *The Canadian Journal of Surgery*. Acceptance is subject to the understanding that they are submitted solely to this Journal, and will not be reprinted without the consent of the author and the publishers. Acceptance or rejection of contributions will be determined by the Editorial Board. As space is available, a limited number of case reports will be published. Articles should be typed on one side only of unruled paper, double-spaced, and with wide margins. The author should always retain a carbon copy of material submitted. Every article should contain a summary of the contents. The Concise Oxford Dictionary will be followed for spelling. Dorland's American Medical Dictionary will be followed for scientific terminology. The Editorial Board reserves the right to make the usual editorial changes in manuscripts, including such changes as are necessary to ensure correctness of grammar and spelling, clarification of obscurities or conformity with the style of *The Canadian Journal of Surgery*. In no case will major changes be made without prior consultation with the author. Authors will receive galley proofs of articles before publication, and are asked to confine alterations of such proofs to a minimum.

Reprints

Reprints may be ordered on a form which will be supplied with galley proofs. It is important to order these before publication of the article, otherwise an extra charge for additional type-setting will be made.

References

References should be referred to by numerals in the text. They should include in order: the author's name and initials in capitals; title of the article; abbreviated journal name; volume number, page number and year. The abbreviations of journal names should be those used by the National Library of Medicine, Washington, D.C., as published in *Index Medicus*. References to books should include in order: author's name and initials; title of book; number of edition (e.g., 2nd ed.); title of publishing house; city of publication; year of publication; page number if a specific reference.

Illustrations

A reasonable number of black-and-white illustrations will be reproduced free with the articles. Colour work can be published only at the author's expense. Photographs should be glossy prints, unmounted and untrimmed, preferably not larger than 10″ x 8″. Prints of radiographs are required and *not the originals*. The magnification of photomicrographs must always be given. Photographs must not be written on or typed on. An identifying legend may be attached to the back. Patients must not be recognizable in illustrations, unless the written consent of the subject for publication has been obtained. Graphs and diagrams should be drawn in India ink on suitable white paper. Lettering should be sufficiently large that after reduction to fit the size of the Journal page it can still be read. Legends to all illustrations should be typed separately from the text and submitted on a separate sheet of paper. Illustrations should not be rolled or folded.

Language

It should be clearly understood that contributors are at full liberty to submit articles in either English or French, as they please. Acceptance will be quite independent of the language of submission. If the contributor wishes, he may submit an informative summary of not more than 300 words in the language other than that in which he has submitted the article. For example, an article in English must carry an English summary and may, if the author wishes, carry a more detailed summary in French.

LE JOURNAL CANADIEN DE CHIRURGIE

Toute communication concernant le Journal devra porter la mention "Le journal canadien de chirurgie" et être adressée à l'Editeur, Publications de l'A.M.C., 150 St. George Street, Toronto 5.

Le journal est publié trimestriellement. Le prix de l'abonnement est de $10. par an ($5. par an pour les médecins qui sont résidents en chirurgie) et commence avec le numéro de janvier de chaque année. Un exemplaire isolé coûte $2.50 et est payable d'avance. (Nous serions reconnaissants aux souscripteurs de vouloir bien ajouter à leur chèque le montant des frais bancaires éventuels).

INSTRUCTIONS A NOS COLLABORATEURS

Manuscrits

Les manuscrits d'articles originaux, de rapports cliniques etc. seront envoyés en deux exemplaires, accompagnés d'une lettre demandant qu'on veuille bien considérer leur publication dans *Le journal canadien de chirurgie*. Ils ne seront acceptés qu'à la condition qu'ils n'aient été soumis qu'à notre Journal et qu'ils ne soient pas réimprimés sans le consentement exprès de l'éditeur et l'auteur. L'acceptation ou le refus des articles soumis relève du Conseil de la publication. Si la place est disponible, un nombre limité d'histoires cliniques pourront être publiés. Les articles seront dactylographiés sur un seul côté d'un papier non ligné, à double espace et avec une large marge. L'auteur devra toujours conserver une copie au papier carbone du texte soumis. Tout article devra être accompagné d'un résumé. L'orthographe sera celle adoptée par le dictionnaire Larousse. Quant à la terminologie scientifique, elle sera basée sur le Dictionnaire des termes techniques de médecine ou tout autre ouvrage de référence sérieux. Le Conseil de la publication se réserve le droit d'apporter au texte les changements qu'il jugerait à propos pour assurer la correction grammaticale et l'orthographe, pour éliminer d'éventuelles obscurités ou pour rendre la présentation conforme au style du *Journal canadien de chirurgie*. Aucun changement important ne sera apporté au texte sans que l'auteur aît été préalablement consulté. Les auteurs recevront avant la publication des épreuves d'imprimerie de leur texte, auxquelles ils sont priés d'apporter le minimum de corrections.

Tirés-à-part

On pourra commander des tirés-à-part sur une formule qui est envoyée avec les épreuves. Il est important de les commander avant la publication de l'article, sous peine de devoir payer un supplément pour une nouvelle composition.

Bibliographie

Les références bibliographiques seront indiquées par des numéros dans le corps du texte. Elles comprendront dans l'ordre: le nom de l'auteur et ses initiales, en majuscules, le titre abrégé du Journal, le numéro du volume, le numéro de la page et l'année. Les abréviations admises pour les noms de revues sont celles qui figurent dans *l'Index Medicus* de la Bibliothèque Nationale de Médecine, Washington, D.C. Les renvois aux livres comprendront dans l'ordre: le nom de l'auteur, ses initiales, le titre de l'ouvrage, le numéro de l'édition (p. ex. 2ème éd.), le nom de la maison d'édition, la ville où elle est située et l'année de la publication; enfin, le numéro de la page s'il s'agit d'un renvoi précis.

Illustrations

Le journal accepte de publier gratuitement un nombre raisonnable d'illustrations en noir et blanc. Les reproductions de clichés en couleurs seront publiées aux frais de l'auteur. Les photographies seront imprimées sur papier brillant, ne seront ni montées ni calibrées et d'un format maximum de 8" x 10". En ce qui concerne les radiographies, nous demandons des copies et *non pas l'original*. On devra toujours fournir un agrandissement de microphotographies. Il ne faut jamais écrire ou dactylographier un texte quelconque sur les photographies. Une légende les identifiant pourra être jointe au dos. Dans les illustrations montrant des malades, ceux-ci ne pourront être reconnus, à moins qu'ils n'en aient donné le consentement écrit préalablement à la publication. Les graphiques et diagrammes seront dessinés à l'encre de Chine sur un bon papier à dessin blanc. Le lettrage devra être écrit en caractères assez grands pour que, après réduction proportionnelle au format du Journal, ils soient encore lisibles. Les légendes devant accompagner les illustrations seront dactylographiées sur une feuille indépendante du texte. Les illustrations ne seront ni roulées ni pliées.

Langue véhiculaire

Il doit être clairement établi que les collaborateurs ont pleine liberté de soumettre leurs articles en français ou en anglais, à leur choix. L'acceptation de l'article sera entièrement indépendante de la langue choisie par l'auteur. Si le collaborateur le désire, il peut décrire le contenu de l'article en un sommaire ne dépassant pas 300 mots et dans une langue différente de la langue choisie pour l'article lui-même. Par exemple, un article écrit en français doit comporter un résumé en français et peut, si l'auteur le désire, être accompagné d'un sommaire plus détaillé en anglais.

BOOK REVIEWS

(See also pages 343 and 410)

ANESTHESIA FOR OPEN HEART SURGERY. Edited by Lillian E. Fredericks and Dryden P. Morse. 80 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1966. $6.50.

This is a short symposium on the conduct of anesthesia for cardiac surgery and the problems of the postoperative period. The discussions of 20 participants were taped and edited.

The book does not begin to cover this interesting and rapidly changing field. The chapter titles are provocative but, with few exceptions, the contents are disappointing. Even the references are completely inadequate. Not even these learned contributors could be expected to develop these topics in so short a space, and they did not do so. The best of the presentations represent the briefest introduction to the topics and the poorer ones represent expressions of opinion and personal preference.

It is difficult to believe that this symposium could be of much value to student or clinician.

BACKACHE RELIEVED THROUGH NEW CONCEPTS OF POSTURE. W. Harry Fahrni. 52 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1966. $6.50.

This is a short treatise on postural low backache, which is presented clearly on good quality paper, with many line drawings and a brief text. It is designed for sufferers from backache.

The author visited a primitive society in India and made interesting observations regarding the habits and posture of the Bhil tribe in their jungle habitat. This study was partially supported by the Canadian Arthritis and Rheumatism Study, British Columbia Division. The conclusions reached are "that these primitive people use their spines in a much more efficient way than we" and that a great deal of the back trouble in our society can be overcome by avoiding the strains of standing, sitting and lying.

The two most serious criticisms of the message in this manual are first, the sweeping attempt to apply postural theories to all human backache and second, the suggestion that the spines of two different races are basically similar. Nothing has been done to establish the occurrence or incidence of degenerative disc disease in the Bhil tribe.

CARDIOVASCULAR PATHOLOGY. Vols. 1 and 2. Reginald E. B. Hudson. 2123 pp. (incl. index). Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1965. $75.00 set.

The author of this scholarly work is a pathologist at the National Heart Hospital and the Institute of Cardiology in the University of London. The book is in two volumes, having a total of over 2000 pages. It is a comprehensive treatise of both congenital and acquired cardiovascular pathology and the text is very readable. Illustrations, although few, are well chosen. Reference lists are extensive and appear following the relevant discussion. An attempt has been made to incorporate pertinent clinical considerations. This is welcome but the results are not of the calibre of the mainstream of the work. The size and price of this book will likely result in its distribution being limited to reference libraries. If the first edition is any indication, this will become a standard text on cardiovascular pathology.

THE CHEST FILM IN MASSIVE PULMONARY EMBOLISM. Daniel J. Torrance. 74 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $8.00.

This monograph deals with the radiological signs of massive pulmonary embolism as manifested on the plain chest radiographs. Twenty-three selected cases are presented and, by means of line drawings and radiographs, the radiological features of these cases are correlated with the gross pathology.

The reproduction of the portable chest radiographs is of exceptionally high quality and illustrates in a satisfactory way the changes referred to in the accompanying text.

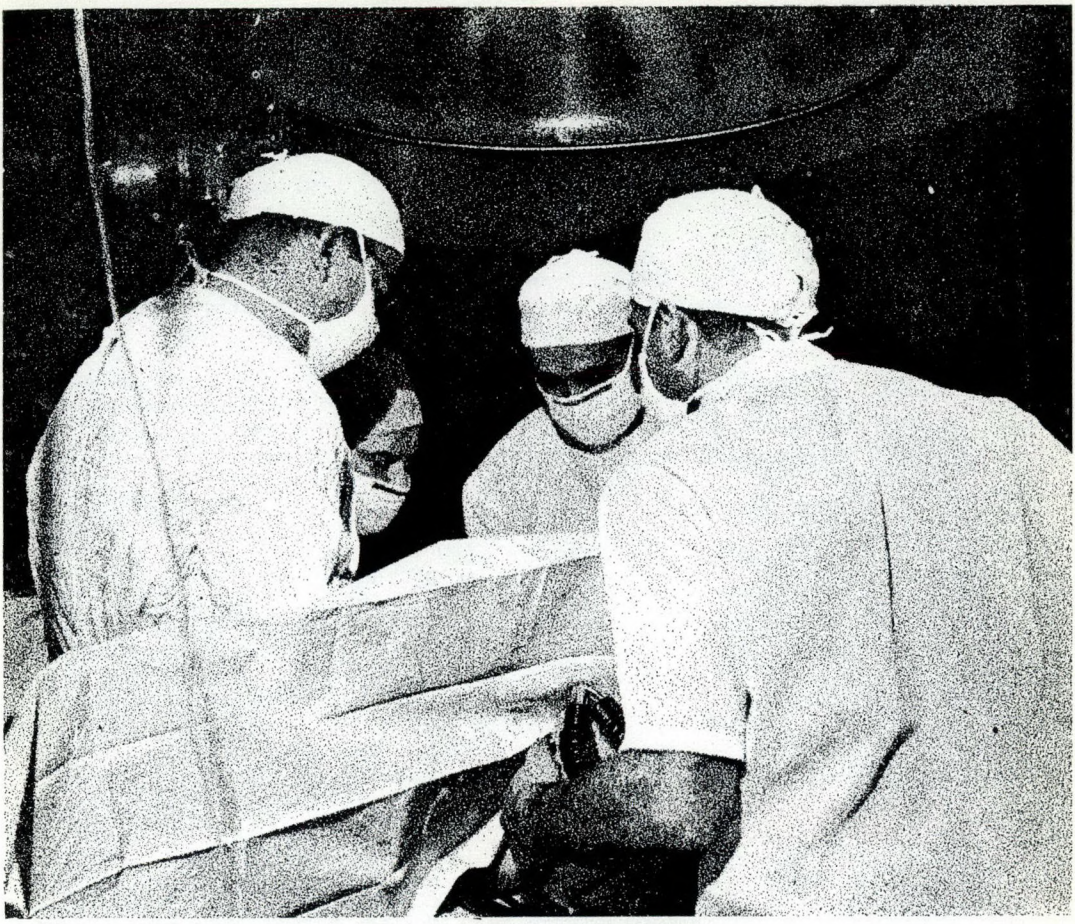
The text is quite lucid but is frequently interrupted by a long series of illustrations, which causes the reader to lose continuity.

Unfortunately, the author gives the impression that the changes described are always present in massive pulmonary embolism and that a radiological diagnosis should be comparatively simple.

The diagnosis of pulmonary embolism is fraught with difficulties and all physicians, particularly interns and surgeons, should find this monograph of interest and practical value.

CLINICAL PATHOLOGY. Clinical Microbiology and the Processes of Disease. J. D. Allan Gray and George Discombe. 857 pp. Illust. Blackwell Scientific Publications, Oxford; The Ryerson Press, Toronto, 1966. $20.00.

Well organized, impressive amounts of modern data are presented in this book "to help students, housemen and general practitioners



"An agent of first choice in obstetrics."[1] Here's why:

Penthrane (methoxyflurane) induction is rapid in obstetric use. [1-4] Very low concentrations (approximately 0.5%) suffice. Such low Penthrane concentrations give an analgesic effect. They relax the perineum, yet don't impair progress of labour, nor unduly depress blood pressure and respiration.

Rarity of vomiting is a particularly striking feature of Penthrane in obstetrics. [1-5] Incidence in typical studies has been under 5%.

An instructive motion picture, "It's a Girl!" gives details on obstetric application. Ask your Abbott man. Or write Abbott, Post Office Box 6150, Montreal, Quebec.

PRECAUTIONS: Suspected liver disease may rule out halogenated agents such as Penthrane. Use only conservative doses of epinephrine, barbiturates, narcotics, tubocurarine, and trimethaphan camphorsulfonate. Guedel eye signs do not apply. Of most value in estimating depth of anaesthesia are blood pressure, respiratory depth, and degree of muscular relaxation.

Professional literature available on request.

PENTHRANE* methoxyflurane

1. Hudon, F., Jacques, A., Clavet, M., Houde, J., Pelletier, J., and Trahan, M., Symposium on Methoxyflurane, Can. Anaes. Soc., J., 10:277, May, 1963, 2. Schubert, F., Keuther, J., Experience with Methoxyflurane in Obstetrical Anesthesia, Proc. Third World Cong. Anesth., 2:25, Sept., 1964. 3. Boisvert, M., and Hudon, F., Clinical Evaluation of Methoxyflurane in Obstetrical Anaesthesia, Can. Anaes. Soc. J., 9:325, July, 1962. 4. Hudon, J., Methoxyflurane, ibid., 8:544, Nov., 1961. 5. Romagnoli, A., and Korman, D., Methoxyflurane in Obstetrical Anaes. and Anal., ibid., 9:414, Sept., 1962.

* TRADEMARK REGISTERED

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to make the best use of the Clinical Laboratory". It is a very concise summary of present-day knowledge on this subject for anyone preparing for Certification or Fellowship in the Royal College of Physicians and Surgeons.

Clinical Microbiology is covered in 414 pages. Besides systemic microbiology and modern immunology it includes discussions of such problems as: pyrexia of unknown origin, cross-infection and prevention of accidental infection among hospital staff.

Processes of Disease, the second part of the book, is divided into three parts: the pathologist's view of the patient, of bodily function and of blood. The discussion of iatrogenic disease, drug toxicity and poisoning is up to date. The relations between cytogenetics and disease are well summarized. In Errors of Metabolism, the latest enzymological advances are included and some humour is also provided: acute attacks of gout are caused by "rich foods, heavy wines and light women". Take your choice! The discussion on water and electrolyte balance is clear and helpful. In Auto-immunity and Disease, the latest information provided by experimental data is related to human disease.

Most chapters have up-to-date basic references. The style is clear with remarkable brevity.

COMPENDIUM OF EMERGENCIES. 2nd ed. Edited by H. Gardiner-Hill. 374 pp. Butterworth & Co. (Publishers) Ltd., London; Butterworth & Co. (Canada) Ltd., Toronto, 1965. $13.50.

This book consists of 22 chapters, written by 24 authors under the general editorship of Dr. H. Gardiner-Hill of St. Thomas' Hospital, London. The subject matter covers the broad field of medical practice and is planned to help the doctor who is "far afield and on his own", as for instance a ship's doctor. It is well designed to serve this purpose as it describes the recognition of each emergency and tells the reader what to do about it. It deals with all the usual emergency situations such as care of the newborn, abdominal conditions, emergencies of the chest, psychiatric emergencies, etc. In addition, it covers less common subjects such as tropical emergencies; hazards of medical practice; air, sea and underwater emergencies; dental problems; and professional negligence.

The book has no claim to profundity, but it does have practicality. It is particularly useful for the newly graduated doctor, the country doctor far from help and consultation, the army doctor, and the specialist who is confronted with a medical problem outside his own field that must be dealt with immedi-

ately. It should be in every emergency department and in the bag of each new intern.

A CURRENT TECHNIQUE OF AORTOILIAC AND FEMOROPOPLITEAL ENDARTERECTOMY FOR OBLITERATIVE ATHEROSCLEROSIS. Jack A. Cannon. 54 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $5.75.

This monograph gives a personal account of the management of patients with obliterative atherosclerosis affecting the aortoiliac and femoropopliteal arterial systems. There are brief notes outlining the author's indications for operative intervention in carefully selected patients. He stresses the need for a conservative approach in patient selection and advises complete preoperative angiographic assessment.

There is a well-illustrated, detailed, step-by-step account of the operative technique of thromboendarterectomy as performed by the author. The technical procedures are described in their entirety and obviously demonstrate the development of a technique by critical reappraisal over years of experience.

The author advises the use of heparin postoperatively for femoropopliteal endarterectomy. This is at variance with many authors as it is felt that the complications of postoperative heparinization outweigh its usefulness.

Operative and long-term results are not discussed but at the outset the author makes it clear that this is not the purpose of his monograph.

This book can be highly recommended for its description of the technique of semiclosed thromboendarterectomy and will undoubtedly be useful to the embryonic vascular surgeon.

PRIMARY HEPATOMA. Edited by Walter J. Burdette. 150 pp. Illust. University of Utah Press, Salt Lake City, Utah, 1965. Available free of charge from National Institutes of Health, Bethesda, Maryland.

The reviewer, as a clinician, approached this monograph on hepatoma with some misgivings, expecting a mass of statistics, complex biochemical discussion and detailed electron microscopic descriptions. Although information of this nature is present it does not overwhelm the reader because it is well interspersed throughout an easily read and fascinating text.

The discussion covers the clinical implications and geographic distribution of hepatoma as well as its relationship to dietary factors, viral hepatitis and parasitic disease. The current investigation of human and animal hepatomas is summarized.

The monograph is recommended for those interested in the clinical and investigative aspects of hepatoma and cancer in general.

THE SCIENTIFIC BASIS OF SURGERY. Edited by W. T. Irvine. 571 pp. Illust. J. & A. Churchill Ltd., London; Queenswood House Limited, Toronto, 1965. $24.50.

This book must be unique in surgical literature. The editor, who is one of the younger school of British surgeons and Professor of Surgery in London University at St. Mary's Hospital, states its aims as follows: "Today a vast mass of basic scientific information is accumulating each year with very considerable application to the serious practitioner of surgery, much of this information being placed in a wide range of specialized scientific journals. For this reason it is becoming increasingly difficult to keep up with the scientific background of surgical practice. This book is an attempt to supply this need for the postgraduate surgical trainee, the practicing surgeon and the surgical teacher."

In achieving this aim he succeeds admirably. The subjects discussed by the contributors he has gathered, all of whom are experts in their respective fields, cover a wide range including gastric function, liver and portal circulation, exocrine and endocrine pancreatic function, pulmonary function, adrenal cortex, repair processes of nerves and numerous others. Each chapter has an up-to-date list of references and there are many excellent illustrations throughout the text. This book is recommended to all surgical trainees who aspire to sit for higher examinations and to all surgeons who are interested in keeping abreast of the basic advances in surgical practice.

SURGERY. Principles and Practice. 3rd ed. Carl A. Moyer, Jonathan E. Roads, J. Garrott Allen and Henry N. Harkins. 1828 pp. Illust. J. B. Lippincott Company, Philadelphia and Montreal, 1965. $19.50.

This is the third edition of what must be now considered a standard textbook of surgery. The essentially physiological approach to the problems of surgery is maintained and this remains the book's strong point. The sections on fluids and electrolytes, hypovolemic shock, burns and intestinal obstruction are all excellent. Particularly good are Dr. Harkins' opinions on what is important and what is not important in the various concepts of etiology and treatment of shock.

This book contains some 1800 pages and is illustrated profusely and well with line drawings, photographs, artists' conceptions and graphs. Each organ and its diseases are ex-

haustively treated and this brings up one of the weak points of the book—too much detail. Although the authors intended it deliberately, to this reviewer there are too many operative details. For example, there is a long description of the technique of radical mastectomy and, incidentally, almost a summary dismissal of simple mastectomy and a rather unscientific lack of any of the mounting evidence in favour of simple mastectomy. One wonders if the routine drainage of all gallbladders should be taught to students or if this is a passing phase of surgery and, therefore, not worthy of mention in a textbook. Is double ligation of small arteries necessary? Is the compounding of stock solutions of electrolytes to be condemned or is it merely a modern logistical advance, which makes therapy easier and safe? Is it not rather the responsibility of the teacher to develop and teach the use of these solutions? These particular opinions, if not perpetuating errors, are perpetuating habits.

This book attempts to cover the broad and expanding field of surgery and, although aimed at medical students, will prove rewarding to residents and clinical surgeons as well. The bibliography of references is monumental and the bibliographic index easy to use and extremely helpful.

Books Received

Books are acknowledged as received, but in some cases reviews will also be published.

The Aetiology of Compressed Air Intoxication and Inert Gas Narcosis. P. B. Bennett. International Series of Monographs in Pure and Applied Biology: Zoology Division. Vol. 31. 116 pp. Illust. Pergamon Press Ltd., Oxford; Pergamon Press Inc., New York, 1966. $6.00.

An Atlas of Orthopedic Surgery. Lewis Cozen. 732 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1966. $16.50.

Backache Relieved through New Concepts of Posture. W. Harry Fahrni. 52 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1966. $6.50.

Clinical Pathology. Clinical Microbiology and the Processes of Disease. J. D. Allan Gray and George Discombe. 857 pp. Illust. Blackwell Scientific Publications, Oxford; The Ryerson Press, Toronto, 1966. $20.00.

Clinical Surgery. General Principles and Breast. Edited by Charles Rob and Rodney Smith. 580 pp. Illust. Butterworth & Co. (Publishers) Ltd. London; Butterworth & Co. (Canada) Ltd., Toronto, 1966. $31.00.

Clinical Surgery. The Hand. Edited by R. G. Pulvertaft. 355 pp. Illust. Butterworth & Co. (Publishers) Ltd., London; Butterworth & Co. (Canada) Ltd., Toronto, 1966. $22.00.

Clinical Surgery. Head and Neck. Edited by Charles Rob and Rodney Smith. 216 pp. Illust. Butterworth & Co. (Publishers) Ltd., London; Butterworth & Co. (Canada) Ltd., Toronto, 1966. $15.75.

Comprehensive Background for Anesthesiology. W. Forrest Powell. 346 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1966. $19.00.

Glaucoma. Epidemiology, Early Diagnosis and Some Aspects of Treatment. Proceedings of a Symposium held at The Royal College of Surgeons of England, June, 1965. Edited by L. B. Hunt. 127 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1966. $2.25. Paperbound.

Gynaecology. J. M. Holmes. 228 pp. Illust. Baillière, Tindall and Cassell, Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1966. $3.25.

Hyperbaric Oxygenation. Proceedings of the Second International Congress, Glasgow—September 1964. Edited by Iain McA. Ledingham. 472 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1966. $12.50.

Ischio-Femoral Arthrodesis. J. Crawford Adams. 112 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1966. $6.25.

Mechanik des Gehens. S. Weil and U. H. Weil. 95 pp. Illust. Georg Thieme Verlag, Stuttgart, West Germany; Intercontinental Medical Book Corp., New York, 1966. DM 16.80. $4.50 (approx.). Paperbound.

Neuro-Chirurgie Infantile. Marc-Richard Klein. 456 pp. Illust. Editions Doin-Deren & Cie, Paris, 1966. 98 F. $21.60 (approx.). Paperbound.

Nouvelle Pratique Chirurgicale Illustrée. Fasc XXV. Jean Quénu. 280 pp. Illust. Editions Doin-Deren & Cie, Paris, 1966. 60 F. $13.25 (approx.) Paperbound.

Obstetrics. 11th ed. Edited by Stanley G. Clayton, Donald Fraser and T. L. T. Lewis. 744 pp. Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1966. $11.75.

Orthopädisches Diagnostikum. Hans U. Debrunner. 125 pp. Illust. Georg Thieme Verlag, Stuttgart, West Germany; Intercontinental Medical Book Corp., New York, 1966. DM 25.00. $6.80 (approx.). Paperbound.

L'Osteochondrose Synoviale Osteochondromatose d'Henderson. M. de Pontville, C. Perreau, F. Cabanne and G. Pipaniol. 132 pp. Illust. Masson & Cie, Paris, 1966. 34 F. $7.50 (approx.). Paperbound.

Primary Hepatoma. Edited by Walter J. Burdette. 150 pp. Illust. University of Utah Press, Salt Lake City, Utah, 1965. Available free of charge from National Institutes of Health, Bethesda, Maryland.

Repair and Reconstruction in the Orbital Region. A Practical Guide. John Clark Mustardé. 382 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1966. $17.00.

Retinal Diseases. Symposium on Differential Diagnostic Problems of Posterior Uveitis. Edited by Samuel J. Kimura and Wayne M. Caygill. 395 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1966. $13.75.

Semiologie Clinique et Paraclinique Générale. Art du diagnostic. J. Ducuing. 837 pp. Illust. Editions Doin-Deren & Cie, Paris, 1965. 120 F. $26.50 (approx.).

Shock. Pharmacological Principles in Treatment. Robert J. Marshall and Thomas D. Darby. 99 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1966. $6.50.

Surgical Approaches to the Neck, Cervical Spine and Upper Extremity. Emanuel B. Kaplan. 246 pp. Illust. W. B. Saunders Company, Philadelphia and London; McAinsh & Co. Limited, Toronto, 1966. $12.45.

Syndromes of Disseminated Intravascular Coagulation. Robert M. Hardaway, III. 466 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1966. $21.00.

A Synopsis of Renal Diseases and Urology. Ashton Miller, N. Slade and H. M. Leather. 276 pp. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1966. $6.25.

Thrombohemorrhagic Phenomena. Hans Selye. 337 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1966. $23.50.

Urinary Diversion. British Association of Urological Surgeons Prize Essay, 1965. Theunis Coetzee. 36 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1966. $1.90. Paperbound.

La Vagotomie dans l'Ulcère Gastro-Duodéno-Jejunal. A. G. Weiss, J. F. Hollender and others. Symposium in Strasbourg, France, May 1964. 430 pp. Illust. Expansion Scientifique Française, Paris, 1966. 113 F. $25.00 (approx.) Paperbound.

FORTHCOMING MEETING

ANNUAL MEETING, AMERICAN ACADEMY OF PEDIATRICS, CHICAGO, OCTOBER 22-27

The 35th Annual Meeting of the American Academy of Pediatrics will be held in Chicago, October 22 to 27, 1966.

The meeting is open to physicians who are not pediatricians. The registration fee is $16 for Academy members, applicants to the Academy, applicants to the American Board of Pediatrics, non-members out of school less than five years and physicians in the Armed Forces. The registration fee for non-member physicians out of school more than five years is $50.

Physicians may write the American Academy of Pediatrics, 1801 Hinman Avenue, Evanston, Illinois 60204, for a preliminary program, and housing and registration forms.

NOTICE

INTERNATIONAL SYMPOSIUM ON ELECTRICAL ACTIVITY OF THE HEART

An International Symposium on Electrical Activity of the Heart is being held May 24 to 26, 1967, in London, Ontario.

This symposium is jointly sponsored by the Ontario Heart Foundation and the University of Western Ontario. It will be of value to those interested in the more recent developments in the study of the electrical activity of the heart, electrocardiography, vectorcardiography and computer analysis.

The invited speakers are from Europe, Asia, United States and Canada.

Accommodation has been reserved at the Symposium Centre, The University of Western Ontario.

For further information please write to Dr. G. W. Manning, Victoria Hospital, London, Ontario.